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THE *TREPONEMA PALLIDUM* IMMOBILIZATION TEST (TPI) AND ITS APPLICATION

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Until Schaudinn and Hoffmann[1] demonstrated *Treponema pallidum* in 1905, the only way of diagnosing syphilis was by its clinical manifestations. Further progress was made in 1906, when Neisser, Wassermann and Bruck,[2] following the complement studies of Bordet and Gengou,[3,4] produced a saline extract of a syphilitic liver which gave positive complement-fixation reactions with known syphilitic sera. The following year, however, Landsteiner, Müller and Pötzl[5] found that syphilitic sera fixed complement in the presence of an alcoholic extract of normal mammalian tissue, and not long afterwards it was found that syphilitic sera flocculated this antigen solution. The antibody so detected was designated reagin. The active component of the antigen used in these serological reactions was found to be a phospholipid.[6]

The establishment of a serodiagnosis for syphilis was a great achievement, but by 1920 there was doubt about the specificity of the tests. In some cases, despite repeated positive reactions, the diagnosis of syphilis could not be confirmed clinically. This observation gave rise to the term 'biologically false positive reaction' (BFP) which is defined as 'reagin activity in sera from non-syphilitic patients'.[5] Numerous diseases such as leprosy, relapsing fever, malaria, typhus, trypanosomiasis, measles and lupus erythematosus may be associated with the occurrence of circulating reagins. Consequently, such sera reacting with the standard antigens for syphilis will be of the BFP type. The sensitivity of the antigens may influence the incidence of BFP reactions; for example, the cruder the lipoidal antigen, the more BFP reactions there are.[7] The active component cardiolipin, discovered by Pangborn[6] in 1941, together with lecithin and cholesterol, forms the purest antigens, but even this reacts with reagins occurring in association with conditions other than treponematosis. Instead of condemning, one should praise this artificial antigen, which has proved to be of outstanding value in the serodiagnosis of treponemal infections. Nevertheless, ever since the discovery of *T. pallidum*, attempts have been made to obtain an antigenic component derived from this organism. The first claim to cultivation was made in 1909, and there have been others since, but no confirmation was ever obtained. The present-day explanation is that the organisms isolated were saprophytic non-virulent spirochaetes which contaminated the lesions allegedly containing *T. pallidum*. In 1948, Nelson[8] took up the problem of cultivating *T. pallidum*. In an attempt to find out the factors influencing the survival of the organisms *in vitro*, he showed that the survival of the anaerobic spirochaetes was considerably improved in a nitrogen atmosphere containing 5% of carbon dioxide. This gas mixture was used in all subsequent experiments. To aid anaerobic conditions a reducing agent, sodium thioglycolate, was incorporated in the survival medium. Substances containing sulph-hydryl groups, glutathione and cysteine, were found to be satisfactory, but only in equimolar concentrations, and their effect was only apparent after 48 hours. Inorganic phosphates and bicarbonates were found to aid survival and, by adjusting the amounts in the medium, it was found that, although a pH range of 6·8 - 7·8 was compatible with good survival, the best results were obtained with a pH of 7·0. These experiments were carried out at a temperature of 30°C, which did not kill the spirochaetes as 37°C did. The temperature was later raised to 35°C, which was found to be optimal.

By adding crystalline bovine albumen—5 g. per 100 ml. of the minimum containing sodium thioglycolate—an 81% survival of the spirochaetes was obtained after 96 hours, and 20% after 144 hours. Pyruvate, citrate and succinate were added to see whether the combination of carbon dioxide and certain intermediates in carbohydrate metabolism would prove beneficial to the spirochaetes. Only pyruvate was of any use.

The *T. pallidum* organisms were grown in the testes of the rabbit, and harvested when a clinical orchitis developed, usually in 7 - 9 days. It was found that the testicular tissue extract was a most important factor in the survival medium. Experiments performed by centrifuging the motile spirochaetes and resuspending them in an identical basal medium without the tissue extract[8] showed no survival after 48 hours.

Here, then, was a medium in which it was possible to keep 50% of the spirochaetes alive from 6 to 8 days. No multiplication was noted during this prolonged incubation, but a lengthening of some of the viable organisms was observed. Motile forms retained their virulence, non-motile ones did not.

Having established a basal medium, Nelson and Mayer,[9] in further investigations, found that the addition of syphilitic serum and active complement to a suspension of motile *T. pallidum* immobilized and killed the spirochaetes. This formed the basis of the TPI test as it is now known.

The TPI test is thus an antigen-antibody-complement reaction. The antigen is a suspension of motile, virulent *T. pallidum* (Nichols's strain) in a special medium. In the presence of active complement, usually obtained by cardiac puncture of guinea-pigs under sterile conditions, the antigen is capable of detecting a specific antibody, immobilisin, which occurs in the serum of practically all syphilitic patients beyond the secondary stage. Occasionally the sera contain other spirochaetocidal components than immobilisin, which could give rise to false conclusions. To detect such substances, e.g. penicillin, a control tube with heat-inactivated

complement instead of active complement is set up in parallel with each serum to be tested.

PRINCIPLE OF THE TPI TEST

(A) Motile spirochaetes + immobilizing antibody→sensitized spirochaetes.

(B) Motile sensitized spirochaetes + inactive complement→motile sensitized spirochaetes.

(C) Motile sensitized spirochaetes + active complement→non-motile spirochaetes.

(D) Motile non-sensitized spirochaetes + active complement→motile non-sensitized spirochaetes.

That immobilisin is different from the Wassermann reagin has been demonstrated by absorbing out the reagin of a syphilitic serum of known titre by flocculation with Eagle antigen.[9] The first absorption brought the reagin titre down from 16 to 2, and it became negative on a second absorption. The immobilisin, as measured by the quantitative TPI test, remained unaffected. Other experiments[10] showed that, in untreated experimental syphilis, the reagin titre, after infection, reached its peak about the 3rd month, then dropped and levelled off at a low titre after 6 months. The titre of immobilisin on the other hand only reached its peak after the 10th month, and remained at a high level. Present information suggests that this high titre persists for many years. The two antibodies also differ in their resistance to elevated temperatures.[9] This was shown by heating 5 separate samples of serum at 60, 64, 68, 70 and 72°C. A progressive decrease in the reagin titre was observed, but no corresponding decrease in the immobilizing activity of the serum.

TEST TECHNIQUE

A pathogenic strain of *T. pallidum* (Nichols) is used in the test and maintained in rabbits by intratesticular inoculation. An inoculum in each testis of 1·0 - 1·5 ml. containing 20 - 30 millions of *T. pallidum* per ml. gives the desired clinical orchitis in 7 - 9 days. To harvest the spirochaetes, the rabbit is killed and the testes removed aseptically from the scrotum, opened, sliced, and introduced into an extraction flask containing the temperature- and gas-equilibrated basal medium. The flask is evacuated and refilled with a mixture of nitrogen and carbon dioxide passed over glowing copper filings to remove traces of oxygen. Gentle agitation for 1 - 1½ hours on a mechanical shaker liberates the spirochaetes from the testicular tissue.

While the spirochaetes are being extracted from the testes, the serum to be tested is added to two meticulously cleaned 10 × 1 cm. pyrex tubes. These tubes are closed by loosely-fitting aluminium caps, which serve to maintain sterility and allow of gas replacement. Active complement is added to one tube, the 'test' tube, and inactive complement to the other, the 'control' tube. The spirochaetal suspension is spun gently for 2 minutes to deposit gross tissue debris and the clear supernatant fluid containing the spirochaetes is decanted, adjusted to contain a fixed number of spirochaetes per volume, and added to the tubes containing the serum and complement. Several controls are put up for each batch, viz.: (1) a tube containing spirochaetal suspension only, (2) a set of serially diluted reference serum, to estimate the sensitivity level of the particular spirochaetal suspension, (3) a known negative serum, to safeguard against hypersensitivity, and (4) a tube containing the suspension with a few drops of methylene blue, to detect any leakage of oxygen into the system. A zero-hour count on the treponemal suspension, which should show at least a 90% motility, is carried out. The tubes are shaken gently and placed in a vacuum dessicator, and the air is replaced 4 times with the mixture of nitrogen and carbon dioxide.

For the inoculation of the rabbit, the testes are transferred from the extraction flask to a sterile urine jar containing 5 ml. of fresh medium. With a sterile glass rod the testes are ground up for about 5 minutes; the supernatant is examined under dark-ground illumination and, if it is estimated to contain sufficient spirochaetes (a minimum of $3{\cdot}0 \times 10^7$ per ml. of suspension), the fluid is drained off and inoculated intratesticularly into a mature rabbit.

The test is read after 18 - 22 hours' incubation at 35°C. A loopful is placed on a grease-free slide, and covered with a clean coverslip. Under dark-ground illumination, 25 successive spirochaetes are counted mentally and the motile ones are recorded on a hand counter.

The treponemal suspension alone (control tube I) indicates the efficiency of the medium in maintaining survival. The motility should be at least 80%. The reference serum controls are read and the percentage immobilization is plotted against the serum dilution on semi-logarithmic graph paper and the dilution giving 50% immobilization is read. From batch to batch this should not vary more than one dilution. A rise in titre may suggest *in vivo* sensitization and, to avoid that, the spirochaetes are not harvested from the testes after more than a 2-day clinical orchitis. The tubes containing the patients' serum, with active and inactive complement, are then read, and the specific immobilization (SI) (defined as the percentage of spirochaetes immobilized entirely as a result of the presence of immobilisin in the serum) is calculated as follows:

$$\%\,\text{SI} = \frac{\%\text{ motile in control tube} - \%\text{ motile in test tube}}{\%\text{ motile in control tube}} \times 100$$

In the interpretation of the results 0 - 19% SI is reported as non-reactive, 20 - 49% as weakly reactive and 50 - 100% as reactive. It is essential for the immobilization that complement has been available throughout the incubation period. Therefore, after the examination of each suspension, the test tubes containing active complement are placed in the refrigerator at 4°C to minimize any further deterioration of complement. A haemolytic system is added to the tubes and incubated at 37°C for 30 minutes, and the occurrence of haemolysis is taken as evidence of excess of complement.

ASSESSMENT OF THE TEST

The advantages and disadvantages of the TPI test may be described by comparing it with the reagin standard tests for syphilis (STS). In a country such as South Africa, where the incidence of syphilis and extra-venereal treponematosis among the non-Whites is high, the STS tests are extremely useful, in that they are sensitive, relatively specific, and inexpensive. The expense of the TPI test naturally precludes it from becoming a routine investigation, but if one is confronted with a positive STS without any clinical or anamnestic support for the diagnosis of treponematosis—be it in White or non-White—the TPI test is of outstanding value because of its high specificity. In such cases, a negative TPI test will exclude syphilis beyond the secondary stage and draw attention to the BFP type of the patient's STS, the nature of which is equally important to establish, but beyond the spheres of the Wassermann and TPI laboratories. This is particularly relevant in pregnant women in order to prevent congenital syphilis. The TPI test is also important in establishing a positive diagnosis in patients who have negative STS but are suspected of having a syphilitic condition such as aortitis, syphilitic lesion of the central nervous system, or congenital syphilis. The advantage of the STS over the TPI test is in the assessment of successful treatment of the patient. A reagin titre will fall completely, but unless treatment is started in the early secondary stage of syphilis immobilisin will disappear so slowly that the reaction normally remains positive for life. Consequently a reinfection could only be established by the reagin tests.

APPLICATIONS OF THE TEST

In 1955 the TPI laboratory was established in South Africa, to provide the clinician with a test whereby he could assess his 'difficult cases'. In addition, an investigation into the

reliability of the STS was desired, and a reassessment of the incidence of treponematosis in the Bantu in the light of modern serology became possible. Some 900 apparently healthy, adult Bantu, partly from Bechuanaland and partly from a township near Johannesburg have been examined with the TPI test. Although the study has not been completed as yet, the preliminary results suggest that at least 33% of this group are or have been suffering from treponematosis. The STS have been found to be comparatively specific but about 25% less sensitive than the TPI test.

Studies undertaken in association with the Westfort Leper Institution have shown that in a group of about 350 leprosy patients the incidence of syphilis was about 25%. The BFP reactions occurred only in the lepromatous type of leprosy but not in the tuberculoid. The BFP was observed to fluctuate on repeat tests and, in an attempt to correlate this fluctuation with clinical findings, a group of syphilitic and non-syphilitic patients based on the TPI test are being examined serologically and clinically at regular intervals. This work is still in progress and it is hoped that some information will be forthcoming about the nature of reagins produced in conditions other than treponematosis.

SUMMARY

The technique of the *Treponema pallidum* immobilization test is reviewed; attention is drawn to the increased specificity and sensitivity of the test as compared to the standard tests for syphilis, and its application in clinical medicine is outlined.

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SURGERY IN OTOSCLEROSIS WITH A REFERENCE TO SUCCESSFUL FENESTRATION OF THE STAPES FOOTPLATE

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The earliest operations for otosclerosis since the introduction of aseptic surgery have been credited to a German otologist, Kessel, who in 1876 removed the tympanic membrane, incus and malleus; the results were not encouraging. Boucheron (1888) and Miot (1890) were the first to perform stapes mobilization, but the operation was abandoned because it was thought to be both useless and harmful. The principles and technique of the fenestration operation were mainly developed by Hölmgren (1916), Barany (1924) and Sourdille (1932), from whose work Lempert devised the present-day one-stage operation for fenestration of the lateral semicircular canal. The results of this operation have been good, and it was the basis of the accepted method of surgical treatment of otosclerotic deafness until 1952, when Rosen[1] published a report which revived interest in stapes mobilization. The results of Rosen's operation and subsequent modifications, including fenestration of the stapes, have been very encouraging.

It is with reference to this operation of fenestration of the footplate of the stapes that the following case is reported:

CASE REPORT

H.R., Coloured male aged 30 years, attended the out-patient department of the Livingstone Hospital on 4 August 1958. Ever since commencing work as a garage attendant 8 years previously, he had noticed a gradual deterioration in his hearing. More recently he had developed noises in the ears. His previous history and family history were negative.

He was found to be an intelligent man, speaking in a soft voice. On general examination no abnormality was detected. Both tympanic membranes were normal and mobile. Weber's sign equivocal. Rinnie's sign negative in both ears. Air conduction

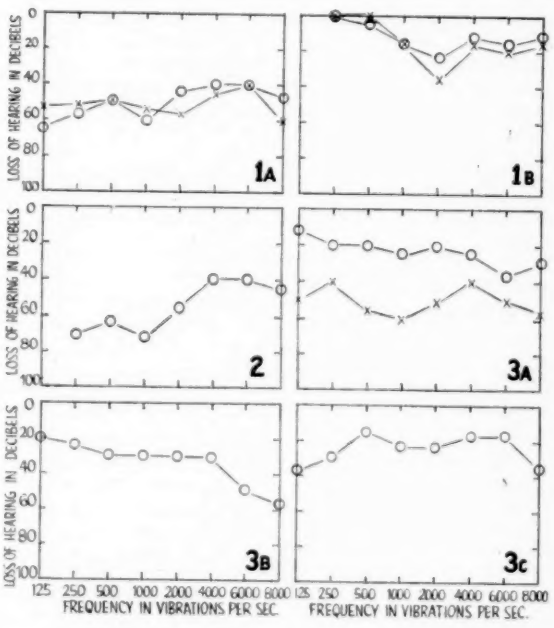

Fig. 1. Audiogram, August 1958. A = air conduction. B = bone conduction. Crosses = left, circles = right.

Fig. 2. After mobilization. Audiogram (right ear), air conduction. November 1958.

Fig. 3. After footplate fenestration. Audiogram, air conduction. A = on 13 January 1959. B = on 19 January 1959. C = on 16 March 1959. Crosses = left, circles = right.

diminished in both ears. The audiogram is shown in Fig. 1 (A and B) for air conduction and bone conduction in each ear.

Bilateral otosclerosis was diagnosed, and on 18 September 1958 a stapes mobilization was attempted on the right ear under a local anaesthetic. The stapes was approached by raising a post-tympano-meatal flap and exposing the middle ear. After manipulations at the stapes neck and incudo-stapedeal joint, the flap was replaced and the ear packed with vaseline gauze. The surgeon did not feel convinced that he had successfully mobilized the stapes. The patient was discharged from the hospital on 21 September.

He was seen in the out-patient department again on 25 September. On this occasion he presented with an acutely inflamed auditory canal and a small posterior perforation of the right tympanic membrane with a pulsating discharge visible in the perforation. He was treated with achromycin (250 mg. *q.i.d.*) for 4 days and local decongestant and cleaning methods were instituted. On this treatment his ear cleared up completely and he was discharged.

He returned to the out-patient department again in November. The right tympanic membrane had healed and the auditory canal appeared quite normal. His hearing showed no improvement. The audiogram (air conduction) on the right ear is shown in Fig. 2.

The alternative lines of treatment were either to do a formal Lempert fenestration of the lateral canal or, assuming the previous surgeon had failed to mobilize the stapes, to make a second attempt at mobilizing.

On 5 January 1959 the middle ear was again exposed by raising a post-tympano-meatal flap. The middle-ear structures were easily identified and it was seen that both crura of the stapes had been fractured. Apart from abandoning the operation there appeared to be no alternative to fenestration of the stapes footplate. Accordingly this was undertaken, and an opening of about 1 mm. square was made in the footplate of the stapes near the posterior crus. All blood clot was removed and a few drops of hydrocortisone introduced into the tympanic cavity. Throughout the operation magnification of ×6—16 was used. The tympano-meatal flap was replaced and the canal packed with tulle gras strips.

The immediate post-operative period was quite uneventful except for mild giddiness during the first 48 hours.

Removal of the ear plug a week later presented a dramatic scene with the patient exclaiming that he could not stand the noise.

An audiogram done on 13 January showed a marked improvement in the hearing (air conduction) of the right ear (Fig. 3). There was some depression of hearing a week later, as can be seen from Fig. 3B.

The patient was discharged on 21 January. On 16 March 1959 an audiogram examination showed that the improvement first noted 2 months previously had remained unaltered (Fig. 3C).

DISCUSSION

It is apparent that clinical otosclerosis presented a great problem to the early otologists, whose failures were probably due not only to a lack of knowledge of the physiology of hearing, but also to the poor surgical technique applied and the absence of aseptic measures. Nevertheless the results of the efforts of early operators and experimenters served to form a basis for those who followed.

It was not until Hölmgren published the results of his work that the surgical treatment of otosclerosis began to advance rapidly. To Lempert goes the honour of the present-day operation of fenestrating the lateral semicircular canal. This appeared to be the only method of surgical treatment until 1952, when Rosen revived the operation of mobilizing the stapes, an operation previously attempted by Miot and others but abandoned because of the poor results produced.

This seemed to be the logical way of treating deafness due to an ankylosis of the stapes, and the Lempert operation to a great extent became the line of treatment only in those cases where the stapes mobilization had failed. The procedure of fenestration of the footplate of the stapes has been mentioned as being a method of treatment in cases where mobilization of the stapes had failed. In the case reported in this article, failure was due to fracture of the crura, and the result of subsequent footplate fenestration was gratifiying. A second case of failed stapes mobilization has since been treated by fenestration of the stapes footplate, and again the result has been satisfactory. If further experience shows this operation to be workable and to produce reasonable results in cases of stapes ankylosis where mobilization is impossible, one feels that it would be the logical procedure to adopt in preference to the Lempert operation.

Not only does it appear to be the best site for creating a fenester, but is will also eliminate so many of the difficulties that arise after a formal Lempert operation, when the cavity constantly requires attention in order to keep it dry and clear of wax. In addition the Lempert operation is a far more formidable procedure and certainly more bloody.

In the case described the patient was detained in hospital for an unnecessary length of time because he was an up-country resident. In this case, as well as the one done subsequently, the patient could have returned to duty within a week or 10 days after the operation. This is naturally an important factor at any hospital where people cannot afford to be detained for any great length of time. The patient who has been subjected to the Lempert operation is expected to pay frequent visits to the clinic for several weeks afterwards. Where distances between home and clinic are great, this may be very costly.

One feels that the case of clinical otosclerosis which warrants surgery should be treated by mobilization of the stapes which, if it fails, could be supplemented by a fenestration of the stapes footplate at the same operation. This approach is surely more logical than to abandon the operation and subject the patient to another anaesthetic at a later date in order to perform a fenestration of the lateral semicircular canal.

SUMMARY

A case of failed stapes mobilization is reported which was successfully treated by fenestration of the stapes footplate, and the instillation of hydrocortisone drops into the middle ear. A brief discussion on the advantages of this procedure is given.

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EDITORIAL : VAN DIE REDAKSIE

THE SOCIAL MISFIT

The word 'psychopath' has become part of ordinary language. In the law courts doctors frequently plead psychopathy as a defence against criminal charges, but in many cases they succeed only in alienating the fourt from their patient because of the vagueness of the concept and, in drawing derisive comment from the Bench, bring psychiatry into disrepute.

In popular speech the term 'psychopath' conveys implications of low moral standards and often of criminality. Psychiatrists have found it virtually impossible to agree on a definition of 'the psychopath'; to state that although he cannot define the term, every experienced psychiatrist can recognize a psychopath when he sees one, is special pleading which is inadmissable. The 'psychopathic tenth' of our community, who break laws, require social support, and offend against the codes of the community, have become the concern of too many lawyers, social workers, doctors and other social agents to be left so obscure. Psychopaths are very real and are common enough to affect almost everybody. To discard this tarnished word and utilize another less emotive term such as 'sociopath', still leaves us with the tesk of medically understanding the people who have personalities which deviate from the average and who, although not sick, cannot fit into our social organization.

In his famous German classic,[1] first published in 1923 but only now translated into English, Professor Schneider expressed views on the concept of psychopathy which remain important. Three distinct elements make up one human individual: intelligence, personality, and the physical organism (soma). An individual's personality is abnormal (and in scientific usage 'abnormal' should imply deviation from the average—whether good or bad being immaterial to the issue) when there is an excess or deficiency of certain personal qualities. The saint, the poet and the criminal are all abnormal in the sense that they fall outside the range of average personality. Schneider therefore conceives that all persons of note may, in this scientific sense, be classed as abnormal personalities. Abnormal personalities are very numerous, but fall into two well-defined groups; those who suffer personally because of their own abnormality, and those who make the community suffer because of it.

Is psychopathy an illness? The social misfit is not ill or sick in any customary sense; his failure to meet the requirements of a given society cannot be equated with any disturbance of bodily function. No morbid change in the body has been demonstrated in psychopaths. Schneider considers the term 'illness' altogether inappropriate for psychopathic personality, and also such catchwords as degeneration or impoverished stock.

People who have suffered from encephalitis lethargica may show residual mental disturbance (usually undirected hyperkinetic activity, which from the outside may be mistaken for meaningful restlessness or motivated tension). In the post-encephalitic pseudo-psychopath the restless drive is blind; the behaviour is not expressive of symbolic emotional content.

Schneider, after having pointed out that there is no brain disorder in psychopathy, asserts that the primary causation is of a genetic nature. Psychopathy is a biological variation. This emphasis on heredity has been much disputed by later writers. Schneider ascribes no more than minor effects to personal history, education and life events. Interested not in interpreting but in observing, his real contribution to this field lies in his description of psychopathic personalities. He attempts no rigid classification, holding that the human personality cannot be labelled diagnostically. 'The same label does not always denote the same psychological situation. For example, people may be "depressed" in more senses than one'.

While he insists that 'we ourselves have never maintained that the psychopathies are to be ascribed wholly to hereditary disposition', Schneider in the main does appear to regard the psychopath as having an inborn disposition, from which stems his vulnerable personality. The psychopath's characteristic constitutional anomaly is exhibited by him as behaviour with some relative *continuity*. For example, those abnormal personalities, the hyperthymes, who show undue good humour and optimism, remain so throughout life. The affectionless psychopaths, who show emotional blunting, are probably equally unvarying. In other types of abnormal personality there may be less continuity. Insecure, attention-seeking psychopaths (hysterical personalities) may, in later life, lose their need to drag their egos into the centre of every situation they encounter. In losing their heightened emotional response, their delight in novelty and their gushing enthusiasm, they become better adjusted persons. Personality traits are moulded by experience; some are weakened with maturity, some reinforced. The traits of insecurity, depression, self-concern and hypochondria are all modifiable.

Schneider's pen-portraits of psychopathic people and the way they behave are of great medical relevance. His phenomenological descriptions enable the doctor to recognize a psychopath when he sees one, and to assign the psychopathic individual to a particular category.

Research has extended our understanding of psychopathy beyond superficial clinical description by a detached medical observer. Contemporary psychiatry has developed methods for discovering the *motivation* of behaviour, and genetic explanations of causation of psychopathy are considered inadequate. A source contributing rich information has been psychoanalysis. A searching study of the introspections of psychopathic persons on the psychoanalytic couch has demonstrated in what way the conscience of the psychopath deviates from the normal. The psychopath's conscience is a poor instrument which fails to inhibit the occurrence of socially irresponsible behaviour. The sense of guilt does not develop adequately, and in consequence the psychopath lacks self-critical capacities.[2] Bowlby[3] has demonstrated the effect of separation from the mother as a cause of delinquent behaviour in later life, and the 'affectionless characters' of children growing up in orphanages is amply documented. Sociological evidence[4] suggests that psychopathy

('the acting-out disorders') is commonest in the lower socio-economic levels. Certain cultural influences can, however, prevent the development of psychopathic tendencies. In a certain Anabaptist religious sect in which self-expression and personal ambition is taboo, the Hutterites, antisocial psychopathy is not seen.[5]

In most societies the problem of psychopathy is troublesome to the community, to relatives and to doctors attempting to aid these emotionally-handicapped people. In South Africa there are no separate institutional facilities for psychopaths who are accepted as such by the courts on the evidence given by doctors called as expert witnesses. Psychopathic offenders often go to gaol although their psychiatric disability is recognized; others are admitted to mental hospitals, where they are miserable, a nuisance to the hospital authorities, and a disturbance to the mentally-ill patients.

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PSIGOPATIESE GEDRAG

Een van die moeilikste geneeskundige-maatskaplike vraagstukke om te hanteer is die probleem van daardie ongevoelige groep mense na wie dikwels as psigopate verwys word. Hulle is die mense wat nie kan aanpas by die normale gangbare waardes van die samelewing waarin hulle leef nie en wat om hierdie rede nimmereindigende verdriet en smart en ellende veroorsaak.

Daar is baie redes waarom dit moeilik is om hierdie mense te verstaan. In die eerste plek is daar gewoonlik niks met hulle verstand as sodanig verkeerd nie. Heel dikwels is hulle selfs begaafde en besondere knap mense. Hulle maak ook gewoonlik, oppervlakkig beskou, 'n baie goeie indruk omdat hulle dikwels hulle woord baie goed kan doen en wel ter tale is. Hulle ly ook nie aan die een of ander spesifieke geestesversteuring wat ons kan omskryf nie.

Die persoonlikheidstrekke van psigopatiese persone val egter in duidelike ‚afwykings'-patrone. En die persoonlikheid en lewenstyl van hierdie soort persone sou waarskynlik op die beste manier as volg beskryf kon word: 'n psigopaat is iemand wat van kleins af 'n volslae gebrek toon aan die algemene gangbare waardes van eerlikheid en betroubaarheid waarop die gewone lewensgang van die groep waaraan hy behoort, berus. Hy kan hierdie gebrek nie aanvul nie omdat hy nie leer deur ondervinding nie; ook het tug en strafmaatreëls geen uitwerking op hom nie. Hy sien net sy eie belange raak, hy is uiters selfsugtig en 'n opperste egoïs en hy sal geen morele oorweging ontsien om sy eie doel te bereik nie. Hy ken geen berou nie. Wet en orde en sedelike oorwegings beteken vir hom slegs die belemmering van sy eie nooit-vervulde strewe na botviering en persoonlike gewin. Die belangrike vraag, wat ons betref, is natuurlik of psigopatie 'n siekte is, of dit berus op aangebore eienskappe, en of dit basies die gevolg is van skewe aanpassingsreaksies. In sy klassieke werk oor hierdie onderwerp, wat reeds al in 1923 verskyn het maar nou eers in Engels vertaal is, dui professor Schneider[1] aan dat hy van mening is dat die grondliggende faktor by die psigopaat van 'n genetiese aard is. Die feit dat hierdie opvatting van professor Schneider sedertdien gekritiseer is, doen nie veel afbreuk aan die waarde van sy boek nie, want die groot verdienste van sy werk lê daarin dat hy psigopatiese persone en hulle gedragspatrone op 'n manier beskryf het wat nou heeltemal klassiek geword het. Vir almal wat in hierdie probleem belangstel, en dit geld veral vir geneeshere en maatskaplike werksters, sal die lees van hierdie boek van groot waarde wees.

As gevolg van uitgebreide navorsingswerk in die moderne psigiatrie wat nie net geïnteresseerd is in patrone van gedrag en entiteite van siekte nie, maar ook in die motivering van gedrag, het ons insig en kennis van hierdie probleem ontwikkel en uitgebrei. Maar, ten spyte hiervan, weet ons nog nie genoeg om ons in staat te stel om hierdie probleem op 'n bevredigende vlak te hanteer nie. Meer navorsingswerk moet nog gedoen word en terselfdertyd moet die probleem van psigopatiese gedrag op so 'n manier hanteer word dat dit die samelewing beskerm teen uitbuiting en dat vraagstukke in hierdie verband tog op 'n beskaafde manier benader kan word. Om hierdie rede is dit noodsaaklik dat ons moet aandring op die oprigting van spesiale inrigtings waarin psigopatiese persone behandel en bestudeer kan word. Op die oomblik dryf hierdie persone na die tronke en na inrigtings vir geestesversteurdes waar hulle eintlik grootliks misplaaste mense is en waar hulle teenwoordigheid negatiewe faktore vorm ten opsigte van die hantering en behandeling van die nie-psigopatiese inwoners van die tronke en van geestesversteurde pasiënte.

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PULMONARY EMBOLISM

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Pulmonary embolism is a common disease and because of its often serious consequences is deserving of study. It has been estimated that in the USA over 33,000 persons die annually as a result of pulmonary embolism and at least 6% of post-operative deaths are due to this cause.[1] So common is the condition that it is held to be the commonest pulmonary disease found in a general hospital.[2,3]

Post-operative embolism is somewhat commoner in the presence of obesity, anaemia, cardiac disease, varicose veins, previous thrombophlebitis, severe infections and carcinoma. Trauma either to muscle or skeleton, fractures, and burns, are among the predisposing causes of thromboembolism as well.

The risk of embolism is proportional to the severity of the surgical procedure; the ratio being lowest after surgery for hernia and chronic appendicitis and highest after gastrectomy, cholecystectomy, splenectomy and hysterectomy.

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AETIOLOGY

Venous Thrombosis

The commonest cause of pulmonary embolism is venous thrombosis, in which pulmonary embolism is a complication in 15% of cases.[4] The loosely attached clot of phlebothrombosis may readily be detached by unaccustomed exercise such as getting up after prolonged recumbency, or straining at stool. The more firmly attached clot of thrombophlebitis is less dangerous, but a relatively safe superficial thrombophlebitis may often be associated with a dangerous deep phlebothrombosis.[2]

A common type of traumatic phlebitis encountered in hospital practice is that due to repeated venipuncture, to prolonged insertion of polythene tubes in veins, and to the intravenous administration of agents such as hypertonic glucose and saline solutions and mercurial diuretics.[5]

The site of venous thrombosis is most commonly in the veins of the calf. A clot in the thigh usually involves the whole femoral vein and may end at the junction with the profunda vein but more commonly extends to the bifurcation of the common iliac. Occasionally the common iliac may be involved as the result of direct spread from the pelvic veins, which are frequently involved in pelvic surgery.

Thrombosis resulting from stasis is a known hazard of prolonged recumbency. Beware of the case that has languished for a long period in a non-surgical ward without exercise and physiotherapy and then is transferred to a surgical unit for a major operation. Here surgery may be followed by an acute embolus within a day or two.

Cardiac Disease

Pulmonary embolism is far commoner in patients with cardiac disease than in the general population. Primary pulmonary thrombosis rather than embolism may develop in cardiac patients, particularly in cases with mitral disease. But, unless some predisposing factor such as pulmonary hypertension is present, primary pulmonary thrombosis is rare. In cardiac failure, circulatory stasis frequently leads to venous thrombosis and pulmonary embolism. The onset of auricular fibrillation in mitral stenosis, or a rapid return of normal rhythm with drugs such as quinidine, may result in pulmonary embolism. Here the embolus arises from a thrombosis situated in the auricular appendix or right atrium. The fibrotic plaque in the right ventricle as a result of myocardial infarction may also lead to thrombosis and embolisms.

PATHOLOGY

Most commonly the embolus is small and frequently multiple. With a large embolus which more or less occludes the pulmonary trunk, the effects on the heart dominate the scene, resulting in acute cor pulmonale.

The lower lobes of the lung are involved most often, the right side more frequently than the left. It is seen less commonly in the mid-portion and rarely in the upper portion. This is explained by the fact that the pulmonary arteries to the upper and middle lobes leave the main pulmonary artery at an acute angle. The infarct is always applied to a pleural surface.

After occlusion of a medium-sized vessel, a haemorrhagic infarct may result, but in healthy lungs the usual result of embolism is incomplete infarction with ultimate complete resolution. Complete infarction is seen more commonly where embolism complicates cases with pulmonary hypertension or congestion, and here healing will be by fibrosis.

The embolus causes congestion and oedema, which is most marked at the pleural surface. In an incomplete infarction, the resultant anoxia causes some alveolar haemorrhage without destruction of the wall, and the ultimate result is complete resolution. In complete infarction, the ischaemia causes destruction of the alveolar walls with resultant haemorrhagic necrosis and later fibrosis.

It has been demonstrated in experimental animals that 60% of the total pulmonary circulation must be obstructed before the blood pressure falls and the right ventricle is embarrassed, and probably 80% before death is likely.[6] Acute circulatory arrest after a massive embolism totally occluding the pulmonary arterial trunk results in sudden death. A less total obstruction (60 - 80%) will cause acute circulatory failure and severe shock. The obstruction to the right ventricular flow reduces the blood return *via* the lungs to the left ventricle and results in a fall of ventricular and systemic pressures. The fall in aortic pressure may reduce the coronary flow and this may simulate a cardiac infarction. In cases with disease of the coronary artery, actual coronary thrombosis may result.

CLINICAL FEATURES

Embolism occurs most commonly 8 - 14 days after surgery. With a massive embolus the essential dominating picture is one of cardiac embarrassment and collapse. With a smaller embolus the common findings are chest pain, haemoptysis, pyrexia, tachycardia and leucocytosis.

The pain is pleuritic; it occurs in the majority of cases because the pleural surface is always involved. In a few cases the pain may be anginal in type, when, in some instances, pleuritic pain may also be present but may be overshadowed by the more severe precordial oppression. A careful history may thus provide the lead to the correct diagnosis.[5] Haemoptysis is characteristic of embolus but occurs in less than 50% of cases and may not be present at the onset. It is frank blood mixed with clear sputum, which differentiates embolism from the rusty infected sputum of pulmonary infection. It is not to be expected in the early stages of incomplete infarction where there is only congestion and oedema. Pyrexia is almost invariably present; it is not influenced by antibiotics but often responds to anticoagulant therapy. The temperature may rise as high as 103°F and last for a few days. A rise in temperature may precede the pulmonary incident as a reflexion of a pre-existing thrombophlebitis.

Other less common features are dyspnoea, cyanosis and jaundice. Dyspnoea may be marked in the large embolus and may be associated with cyanosis. Jaundice as a result of haemolysis of blood in the infarct is very rare indeed except in patients with large pulmonary emboli and cardiac failure. Hepatic dysfunction in these cases may be the more likely cause.

Physical signs are usually inconspicuous in the incomplete infarction. A pleural friction rub is not commonly detected, for it may take 24 hours to develop. Frequent and careful auscultation of the chest is necessary to elicit this important sign, which is often faint and localized to a small area. Exquisite local chest tenderness to palpation may also be elicited. This occurs in pneumonia patients with pleuritic pain but is said to be commoner with em-

bolism.[5] It is apparently produced by spasm of the intercostal muscles secondary to the inflammation of the pleura. Other features are slight dullness on percussion, impaired air entry and a few crepitations localized to the affected area. In the complete infarct the signs are more obvious—the dullness is more marked, there may be bronchial breathing as well as crepitations, and often there are signs of a pleural effusion.

Repeated careful examination of the extremities for evidence of tenderness along the course of the deep veins is necessary in every case. The signs of venous thrombosis may only appear a week after the attack of pulmonary embolism. With prompt diagnosis of pulmonary embolism and the early institution of anticoagulant therapy, late thrombosis will appear less often.

ELECTROCARDIOGRAPHY

An ECG is useful to exclude primary cardiac pathology in the case with a large embolus. Coronary thrombosis can be ruled out on ECG except in the rare cases where there is poor coronary flow, which may simulate this condition. In doubtful cases repeated recordings are useful, because in pulmonary embolism there is a rapid return to normal.

The changes seen are the result of altered position of the heart as a consequence of right ventricular dilatation. The main features are the following: Large S deflection in lead I, prominent Q and inverted T waves in lead III, and in some cases inverted T waves in the right-sided precordial leads.

Case Report

F.S., a rather obese European woman aged 44, was admitted to the Johannesburg General Hospital with a twisted right ovarian cist of 4 days' duration. At laparotomy the cyst was found to be grossly haemorrhagic and the ovarian and uterine vessels appeared to be thrombosed. The cyst and ovary were removed and the patient made a good initial recovery from the operation. Next day, about 12 hours after operation, she suddenly became collapsed, complaining of dyspnoea and substernal discomfort. She was deeply cyanosed and dyspnoeic. Her extremities were cold and the blood pressure could not be recorded. No evidence of deep-vein thrombosis could be elicited in the extremities. An ECG taken shortly after the pulmonary embolus occurred showed changes consistent with an acute massive pulmonary embolus (Fig. 1). It also showed features of coronary ischaemia but another ECG, taken after the patient had been resuscitated with levophed and a blood transfusion, showed no features of coronary ischaemia (Fig. 2). The patient recovered after treatment in the manner described below.

RADIOLOGICAL FEATURES

It is important to realize that the chest radiograph may look normal after embolism. This is true of major emboli which do not produce infarctions and of minor emboli which may or may not produce small infarctions at the lung periphery. Some patients may have showers of emboli for years and eventually develop chronic cor pulmonale without radiographic shadows indicative of infarction. The changes produced by infarction may only become visible after a period varying from hours to days and, unless repeated radiographs are taken, the diagnosis may be missed. Hazy densities may appear and then, after 24 - 48 hours, disappear. These represent alveoli filled with blood, with rapid resolution in the absence of actual infarction.

Another difficulty is that the patient is often too ill for other than bedside X-rays and these are all too frequently of poor quality. Every effort should be made to obtain good X-rays, even if it means moving the patient to the radiology department in his bed.

Lastly the interpretation of findings may be difficult because the signs may mimic other conditions. Practically any shape of opacity can be caused by an infarct. With acute cor pulmonale, lateral and oblique radiographs may show a suggestive bulge of the right ventricle. In cases where there are no pulmonary or pleural changes, the mid-right (Mr) diameter of the heart may lead to the diagnosis of major embolus.[7] The Mr diameter of the cardiac silhouette is the greatest horizontal distance between the mid-spinal line and the right border of the cardiac silhouette in the frontal plane. It rarely exceeds 50 mm. in the normal. An Mr diameter greater than 50 mm. tends to be associated with an increased right atrial volume. Other conditions,

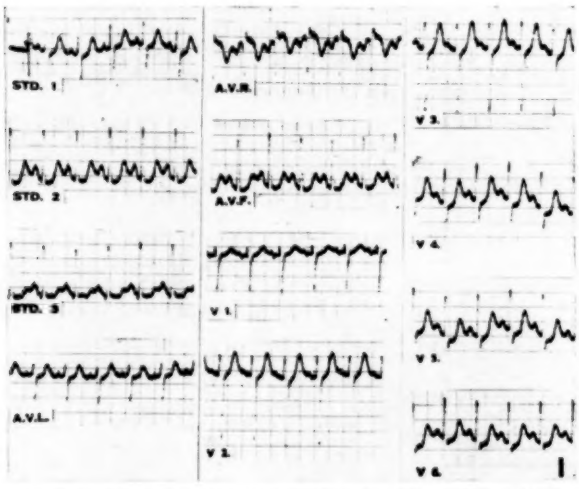

Fig. 1. Case F.S. Note the tachycardia. The standard leads show marked right axis deviation with large peaked P waves in the 2nd standard lead. There is an ST depression over the left anterior chest leads with rather high, peaked and symmetrical T waves, indicating myocardial ischaemia.

Fig. 2. Case F.S. The rate is now much slower. There is still some evidence of right axis deviation. The P waves in standard 2 are still sharp but of normal amplitude. The ST depression previously noted over the anterior chest leads has disappeared and the T waves have returned to normal.

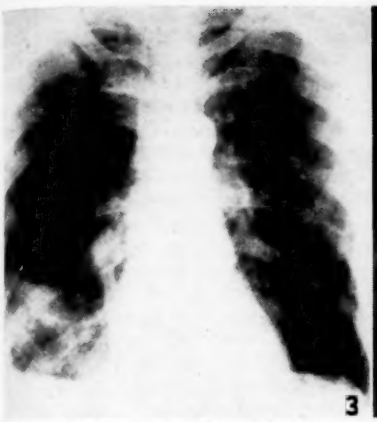

Fig. 3. Case J.S. Chest radiograph in the postero-anterior plane showing a rounded opacity in the base of the right lung. There is also a small pleural effusion at the right base.
Fig. 4. Case J.S. A lateral chest radiograph of the same patient as in Fig. 3, demonstrating the relationship of the opacity to the interlobar pleura (oblique fissure) of the lung.
Fig. 5. Elderly European woman with pulmonary embolism who presented with upper abdominal pain. Chest radiograph showing a markedly raised diaphragm on the right side. The raised diaphragm was found to be immobile (fixed) on screening.

such as pericardial effusion and diaphragmatic hernia, may also increase the Mr diameter and these have to be excluded first.

A pulmonary opacity is to be expected when complete infarction predominates. The triangular shadow which at one time was thought to be the typical finding is rarely encountered. An ill-defined non-homogenous opacity is much more characteristic.[2] If the opacity can be shown to be related to a pleural surface it is then in keeping with a diagnosis of pulmonary embolism. Routine postero-anterior or lateral views sometimes reveal this relation to the pleura but, if not, then careful screening in varying degrees of obliquity may establish such a relationship of an apparently nondescript opacity, seemingly centrally located (Figs. 3 and 4).

Case Report

J.S., a European man aged 58, was admitted for haemoptysis and chest pain. He had once had an amputation of his right leg for 'thrombosis'. On examination he showed no evidence of dyspnoea, cyanosis or jaundice, and no obvious distress. Temperature 98°F, pulse 72 per minute and regular respiratory rate 18 per minute, blood pressure 110/70 mm. Hg. The right leg had been amputated at the mid-thigh level. The left leg showed varicose pigmentation with varicosities of the long saphenous system. Peripheral pulses were present in the leg and there was no evidence of thrombosis of the deep veins. Examination of his chest revealed dullness and crepitations at the right base. A radiograph of the chest showed a right-sided pulmonary opacity and an effusion at the right base (Fig. 3). A lateral radiograph showed the opacity to be related to the pleura of the oblique fissure. (Fig. 4.)

When pulmonary embolism occurs without infarction, the radiograph may show a prominent pulmonary artery and an abnormal translucency of the lung supplied by this vessel, as a result of absence of vascular markings. This is known as Westermark's sign, but in practice it is too difficult to evaluate to be reliable.

The diaphragm is frequently raised on one or both sides as a result of impaired ventilation. This may be the only feature noted in an early X-ray (Fig. 5). Sometimes diminished excursion of the diaphragm is seen on radioscopy; occasionally it is seen to move paradoxically. The cause of this seeming diaphragmatic paralysis is unknown.

Pleural involvement is almost invariable. A small pleural effusion is often found and sometimes may be large (Fig. 3). A minimal pleural reaction of the costophrenic angle with a hump-shaped medial border, known by American radiologists as an 'ice-cube' appearance, is considered diagnostic[8] (Fig. 6).

In incomplete infarction, when the infarct has shrunk to a flat subpleural fibrotic plaque it appears on X-ray as a linear opacity. This is characteristically transient, but it constitutes the most typical finding of all. These lines, sometimes converging, may be the only recognizable residue pointing to the diagnosis (Fig. 7).

DIFFERENTIAL DIAGNOSIS

Post-operative atelectasis may be difficult to differentiate from pulmonary embolism. Atelectasis tends to occur in the immediate post-operative phase, and infarction usually later. Obstruction of a lobar bronchus gives rise to lobar collapse or massive collapse. Obstruction of several smaller bronchi causes a more scattered and patchy form of collapse. With infection the picture eventually resembles lobar pneumonia or bronchopneumonia. The radiological picture is thus either a wedge-shaped or triangular shadow at one base or numerous fluffy opacities, usually basal and often bilateral.

Pneumonia may resemble infarction both clinically and on X-ray; but pneumonia is almost invariably preceded by an infection of the upper respiratory tract and the absence of such illness should lead to the suspicion of embolism. Recurrent episodes involving one lung and then the other —the characteristic picture of embolism—are relatively infrequently due to pneumonia.

Myocardial infarction is the disorder most commonly confused with acute pulmonary embolism. Besides ECG, estimation of the serum-transaminase level is also useful in the determination of the diagnosis. Elevation of the serum transaminase occurs in association with myocardial, hepatic or muscular lesions.

Abdominal catastrophe. Pulmonary embolism has been known to present with severe abdominal pain and muscular guarding simulating true rigidity.[5]

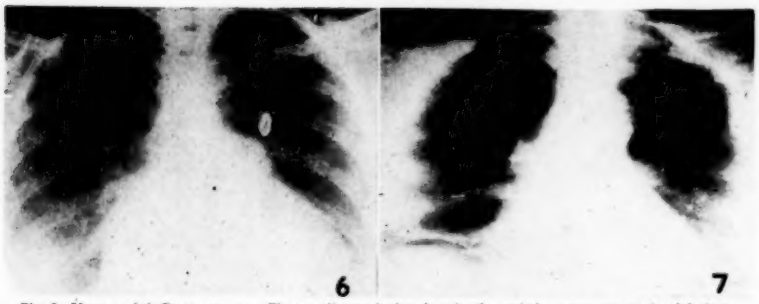

Fig. 6. Young adult Bantu woman. Chest radiograph showing the 'ice-cube' appearance at the right base. Note the enlarged heart due to mitral disease.
Fig. 7. Chest radiograph showing a linear shadow running above the right leaf of the diaphragm.

TREATMENT

Antibiotics are necessary in order to prevent secondary infection after pulmonary embolism. Cavitation has been known to follow pulmonary infarction, and secondary infection may lead to a lung abscess. Cardiac drugs such as digitalis will be necessary in the event of cardiac failure. Cardiac embarrassment in itself, without obvious cor pulmonale, will benefit with digitalis.

Mercurial diuretics are useful to relieve pulmonary congestion and may cause dramatic resolution of infarction.[2]

Omnopon, $\frac{1}{3}$ of a grain, will allay restlessness and anxiety.

Oxygen should be administered if there is breathlessness and cyanosis. Dyspnoea and cyanosis are best treated by propping the patient up, but a precipitous fall in blood pressure with a large embolus is best relieved in the first instance by laying the patient flat and, if necessary, propping up the foot of the bed. This will immediately relieve the effects of cerebral anaemia. For vascular collapse, venesection is considered inadvisable and blood transfusion in small amounts, until the blood pressure has recovered, is now being advised.[9] Drugs such as methridrene or wyamine should be given in the first instance to help restore the blood pressure but one should not hesitate to use levophed (noradrenaline) if the systolic blood pressure falls below 90 mg. Hg. Blood and plasma given with levophed (into a separate vein) will shorten the time otherwise needed by levophed.

Atropine, 1/100th of a grain, and papaverine, $\frac{1}{2}$ - 1 grain, will tend to relieve any bronchial spasm. Where there is any degree of shock, intravenous cortisone (100 mg. of solucortef) should be administered and repeated if necessary.

Anticoagulants should be given in all cases of pulmonary embolism. Their aim is to prevent secondary thrombosis at the site of embolism and to promote canalization of the clot. If anticoagulant therapy is applied early in cases of venous thrombosis, pulmonary embolism almost never occurs. What has to be avoided is inadequate dosage. Heparin is the only agent that can be expected to achieve immediate halting of clot propagation. The oral anticoagulants, or coumarin drugs, act by prolonging the 'prothrombin time' without altering the actual clotting time. Moreover, the response of patients to oral anticoagulants is unpredictable and thus it is difficult to procure a constant effect on prothrombin activity. Therapy should therefore commence with heparin, and the oral anticoagulants should be timed to take over on the 4th - 5th day, by which time clot fixation at the site of venous thrombosis will have been established. The oral anticoagulants are then used to discourage further thrombosis.

Heparin acts directly upon the clotting mechanism by inactivating thrombin. It also halts prothrombin activity and this should be remembered when heparin and oral anticoagulants are used together. The action of heparin is rapid and lasts from 4 to 6 hours according to the dose used. Blood should be taken 1 hour after injection to measure the clotting time. The aim is to produce a clotting time of 15 - 20 minutes, or 3 times the normal. Heparin is best given intravenously, but the deep subcutaneous route is useful when there are vein difficulties, and then pain can be overcome and absorption speeded up by the addition of procaine and hyaluronidase. The recommended adult intravenous dose of heparin is 15,000 units, followed by 10,000 - 15,000 units 4 or 6 hourly, depending upon the clotting time. One should not hesitate to step up the dose if necessary; my own experience is that 10,000 units is frequently inadequate. In massive pulmonary embolism, the initial loading dose should be 25,000 units.[9] During subsequent days, with clinical improvement and when the oral anticoagulants are exerting their full effect, heparin is successively cut to 8 hourly, 12 hourly, and daily; and then stopped.

The oral anticoagulants all have a time lag of 24 - 48 hours or longer before their clinical effects become evident. Patients vary in their sensitivity to the coumarin drugs; they may even vary from day to day. A patient may be resistant to one drug and not to another. The withdrawal of oral anticoagulants after prolonged therapy should be slow, for 'rebound clotting' may follow and in an elderly cardiac this may result in a further coronary occlusion. Dosage is controlled by estimating the prothrombin time. The last heparin dose should be given at least 4 hours before the test, for it also tends to prolong the prothrombin time.

Vein ligation. During the past few years ligation of the femoral vein and the inferior vena cava has to a great extent been abandoned. Experience has shown that after adequate anticoagulant therapy further embolism is rare and operation thus unnecessary. Surgery still has a place in the management of recurrent or pyaemic emboli, or when anticoagulants are contra-indicated as in patients with hepatic renal insufficiency, ulcerative colitis, etc.

Mitral valvulotomy. Where recurrent infarction occurs in the presence of mitral stenosis, it is probable that the infarction is caused not by emboli but by primary thrombosis due to pulmonary hypertension. These cases are often dramatically relieved by mitral valvulotomy.[2]

Pulmonary embolectomy. In 1908 Trendelenberg described his operation for pulmonary embolectomy, but the first successful case was performed by Kirschner in 1924. In 1938 Lewis performed the first successful operation in Great Britain, by which time 11 successful cases had been reported.[10] For all this, most people would contend that patients well enough for surgery are not in need of embolectomy.

Prognosis

The source of the embolus is the uncertain factor in prognosis and so long as this remains, the prognosis is uncertain. The effect on the lungs is the least important because, unless an infarct is extensive, the residual scar will be negligible. The heart is fortunately not embarrassed in the majority of cases, but sometimes recurrent emboli ultimately lead to chronic cor pulmonale.

Prophylaxis

Certain primary principles, such as suitable preparation of the patient for operation, gentle surgical technique, and the prevention of stasis in the veins of the legs during convalescence, are cardinal rules. One should avoid trauma to the limbs on the operating table and, if possible, saphenous vein drips should not be used. The patient in hospital for investigation should not lie idly in bed. Breathing exercises to speed the venous flow should be taught. Early ambulation is also important, and where this is contraindicated leg exercises in bed should be instituted. It has been shown that wearing elastic stockings increases the venous flow from the veins of the legs and reduces the incidence of pulmonary embolism. In a New England hospital where elastic stockings were supplied as a routine measure to a series of 5,467 medical, surgical and obstetric patients over 20 years of age, the incidence of pulmonary embolism was reduced by half.[11] This clearly suggests that elastic stockings should be supplied before surgery to all patients with varicose veins or with a previous history of thrombosis.

SUMMARY

1. A review of pulmonary embolism is presented, with case reports. At least 5% of post-operative deaths are due to this cause. The incidence is in direct proportion to the severity of the operative procedure and conditions such as severe trauma, including burns, are among the predisposing causes as well.

2. Venous thrombosis is the commonest cause of pulmonary embolism. Primary pulmonary thrombosis can also occur in conditions associated with pulmonary hypertension and oedema as seen with cardiac patients. In these cases complete infarction will commonly result whereas, in cases where the lungs were otherwise healthy, incomplete infarction is the rule. The infarct is always applied to a pleural surface.

3. An electrocardiograph is useful, mostly to exclude coronary thrombosis in cases with a major embolus.

4. The X-ray features are presented. A negative X-ray does not exclude pulmonary embolus. The usual picture is a pulmonary opacity related to a pleural surface. The diaphragm may be elevated on the affected side, may be fixed, or may move paradoxically. There may also be a pleural effusion, or an 'ice-cube' appearance at the costophrenic angle. If linear opacities are seen, they constitute the most typical finding of all. With a major embolus the enlarged right border of the heart may be the only clue to the diagnosis. In these cases the mid-right (Mr) diameter of the heart is greater than normal.

5. The differential diagnosis, clinical features and treatment are also presented. Heparin is the anticoagulant of choice in the first instance. The danger with heparin is inadequate therapy, not overdosage.

6. The prognosis as regards the heart and lungs is good but so long as the primary source of the embolus remains, the prognosis as regards further embolization is uncertain. Prophylaxis is discussed and the value of elastic stockings is mentioned.

I am most grateful to Dr. R. Glynn Thomas, of the Department of Radiology, General Hospital, Johannesburg, and to Dr. B. Gollach, formerly of the Department of Medicine, for their asisstance and advice; and I should like to thank Miss M. Tomkins, senior radiographer, for the X-ray photographs and Mr. D. Smith, technician to the Department of Surgery, for the ECG photographs.

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STRONGYLOIDIASIS IN DURBAN

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Strongyloidiasis has always been difficult to eradicate from the body and has tended to be overlooked as a cause of disease in the past. Vogel,[21] for example, thinks infestation by strongyloides worms is relatively unimportant, and fatalities are very rare, while Gelfand[10] considers strongyloides to be quite harmless. In the temperate parts of the world this infestation may also occasionally be found, but its significance may not be appreciated by the clinician; in fact strongyloidiasis may even be missed altogether on routine examination of the patient.

The purpose of this paper is to emphasize the importance of strongyloides infestations and to compare the results of certain specific therapies in these cases.

PRINCIPLES AND METHODS OF STUDY

Fülleborn[8] noted the high oxygen requirements of the larvae of *Strongyloides stercoralis*, and Costello and Grollman[3] demonstrated the marked sensitivity to cyanides of the cytochrome system in *S. papillosus*, a close relative to *S. stercoralis*. It was therefore decided to test the efficiency of potassium-thiocyanate therapy in cases of human strongyloidiasis, hoping thereby to attack the worm during its passage through the body. This drug was chosen for investigation because it was less toxic than a cyanide (being in fact a detoxification product of cyanide in the body); it readily diffuses throughout all tissue fluids and has a generalized effect on all tissues of lowering their metabolism.[5,15]

All the cases were European and Coloured patients from Durban. They were kept under strict supervision in hospital during treatment. Apart from a thorough examination on each patient (including a battery of liver-function tests and eosinophil counts, both being repeated serially throughout the investigation) serum levels for thiocyanate were checked in every case where this drug was given.

Since the strongyloides worm starts ovipositing after about 2 weeks from the time of initial infestation,[17] it was considered advisable to give all specific therapies for a minimum period of a fortnight. All dosages were given according to those recommended in the British Pharmacopoeia (dithiazanine—Partel—tablets were given in the dosage recommended by the manufacturers). Stools were repeatedly examined for larvae after completion of the specific therapy over a period of at least 1 month. A minimum of 6 stool examinations were made, direct smears and concentration methods being employed each time, and no case was considered to be cured if larvae could be found during this post-treatment period. Duodenal aspirates were not examined for larvae, reliance being placed entirely on repeated stool examinations over a long period of time.

Several patients were also infested by other species of worms and had been given various forms of specific treatment (Table I).

TABLE I. CLINICAL ANALYSIS OF CASES OF STRONGYLOIDIASIS

	Race, sex (a)	*Nutrition (b)*	*Other disease (c)*	*Other helminths (d)*	*Therapy (e)*	*Comments*
1.	EF	G	DM	—	nil	No follow-up
2.	EF	M+	—	—	nil	Died (mesenteric thrombosis)
3.	EF	M	—	Tri. Ent.	Hex.	Improved by diet
4.	EM	M+	Emp.	—	Hex.	Improved by diet
5.	EM	G	Pn.	—	Hex. Thi.	Pneumonia resolved No worm cure
6.	CM	M+	B'sis	Ts. Asc.	Thi.	Improved by diet
7.	CF	G	—	—	Thi.	Cured
8.	CF	M+	—	Tri. Asc.	GVo Thi.	No cure Cured
9.	CM	M+	—	Tri. Ank.	Thi. Di.	No cure Cured
10.	CM	M	—	Tri. Asc.	Thi. Di.	No cure Cured
11.	CF	M	—	Ank.	Di.	Cured
12.	CF	M+	Atel.	Tri.	Cort. Di. GVi	Died
13.	CM	G	—	Tri. SM	Di.	Cured
14.	CF	M+	—	Tri.	Di.	Cured
15.	CM	M+	—	—	Di.	Cured
16.	CM	M+	—	Tri.	Di.	Cured
17.	CF	M+	—	Tri. TS Ank.	Di	Cured
18.	CF	G	—	Tri.	nil	No cure
19.	CM	M+	Pn.	Tri. Asc.	GVo	Pneumonia resolved Improved by diet
20.	CF	G	Pn.	Tri.	GVo	Pneumonia resolved Cured
21.	CF	M+	Atel.	Tri.	GVo	Improved by diet

(*a*) C = Coloured. E = European. M = male. F = female.
(*b*) G = nutrition good. M = malnutrition. M+ = severe malnutrition.
(*c*) DM = diabetes mellitus. Emp. = emphysema. Pn. = pneumonia. B'sis = bronchiectasis. Atel. = atelectasis.
(*d*) Tr. = Trichuris. Ent. = Enterobius. Ank. = Ankylostoma. Asc. = Ascaris. TS = *Taenia saginata*. SM = *Schistosoma mansoni*.
(*e*) Hex. = hexylresorcinol. Thi. = thiocyanate. Di. = dithiazine. GVo = oral gentian violet. GVi = intravenous gentian violet. Cort. = cortisone.

RESULTS AND DISCUSSION

Although the majority of the cases showed multiple helminthic infestation, only the effect of therapy on strongyloides is considered in Table I. These effects are mentioned in greater detail in Tables IIA and IIB.

TABLE IIA. RESULTS OF TREATMENT FOR STRONGYLOIDIASIS (NUMBER OF CASES)

Specific therapy	*Apparent cure*	*Failure*	*Total treated*
Dithiazanine	8	1	9
Thiocyanate	2	4	6
Gentian violet orally ..	1	3	4
Hexylresorcinol ..	0	3	3

TABLE IIB. RESULTS OF POTASSIUM THIOCYANATE THERAPY

Case	*Average blood level of thiocyanate, mg.%*	*Comments*
5 EM	4·0	Unsuccessful
6 CM	10·0	Unsuccessful after 21 days
7 CF	6·0	Cured after 18 days' therapy
8 CF	4·0	Cured after 27 days' therapy
9 CM	10·0	Unsuccessful after 18 days
10 CM	8·0	Unsuccessful

In this small series of cases there were 2 deaths, one of which may be directly attributed to heavy parasitization by strongyloides worms. As a cause of death, strongyloidiasis has been reported previously by Grové and Elsdon-Dew[11] from Durban; it cannot therefore be considered as a harmless parasite in the body. It is virtually impossible to assess accurately the degree of infestation among the various populations of Durban, but Elsdon-Dew[6,7] mentions the presumptive rates among Native and Indian communities (see Table III), and suggests that the degree of parasitization is directly related to economic conditions. Galliard[9] also thinks that fatalities from strongyloides infestation are always in patients in poor physical condition.

During the present investigation it was noted that diet alone produced a marked improvement in the patients' general condition even though the infestation had not been eliminated (Table I). Obviously this should be considered only as a general supportive measure and not as a specific form of therapy. It seems unnecessary, therefore, to assume a hyper-virulent change in the strongyloides worm as postulated by Galliard.

The advantages of dithiazanine (Partel) in the treatment of intestinal worms have been discussed by several authors,[1,18,19,22] and have been confirmed in this present series for strongyloidiasis—provided the drug is given in adequate dosage over a long enough period of time. The only failure in this series occurred in a very debilitated patient who died before any therapy could become effective. Partel unquestionably seems to be the drug of choice for the eradication of strongyloides worms.

TABLE III. STRONGYLOIDIASIS AMONG VARIOUS COMMUNITIES (ABSTRACTED)

Authority	*Geographical area*	*Personnel examined*	*Infestation rate %*	*Comments*
Elsdon-Dew[6]	Durban, S. Africa	Indians Africans	8·2 1·67	Hospital cases only
Elsdon-Dew[7]	Durban, S. Africa	ex housing estate	1·0	Lower income groups
Jiménez-Quiros *et al.*[12]	Costa Rica, S. America	Students; male Students; female	1·6 0·6	Random sampling
Kuntz *et al.*[13]	Egypt	Villagers	0·85	Random sampling
Present series	Durban, S. Africa	Europeans and Coloureds	1·0	Hospital cases only

The effects of thiocyanate therapy were disappointing, only 2 out of 6 cases being cured. Thiocyanate, which is potentially poisonous to the patient, had no effect on any other worm. The thiocyanate ion, being readily diffusible throughout the body, must have a direct effect on the migratory stages of the worms in the tissues of the host, but where this actually took place was not determined. During therapy there were no obvious symptoms referable to the lungs or to the liver; there was no marked variation in the eosinophil count; and no abnormal results of liver-function tests could be attributed to this infestation. All larvae excreted in the stools were still viable.

There have been conflicting reports about the value of specific treatments for strongyloidiasis in the literature. Deschiens and Bénex[4] recommend a bismuth-subnitrite mixture as an alternative method of treatment for this condition. Torres[20] and McFadzean and Smithers[14] both consider heterazan and piperazine salts valueless. Corrigan[2] noted a temporary remission of symptoms and death of larvae with hexylresorcinol but that gentian violet (orally or intravenously) was quite unsuccessful in clearing up pulmonary strongyloidiasis. Palmer,[16] on the other hand, agrees that oral gentian violet is useless, but that this drug is a satisfactory therapeutic agent when given intravenously. With the exception of intravenous gentian violet, all these forms of treatment attempt to eradicate the infestation from the gut. Oral thiocyanate, however, may have a limited part to play in attacking the worm elsewhere in the body.

Palmer[16] noted that the eosinophilia may disappear in approximately 25% cases after cure. In the present series, no reliance could be placed on eosinophil investigations because the patients were continually being re-exposed to fresh helminthic infestations once they were fully convalescent and back home; even during the course of treatment there was no change in the eosinophilic response that could be correlated with the change in the clinical condition of the patients. The only adequate criterion of cure is repeated failure to demonstrate larvae of strongyloides; all ancillary investigations are of general clinical value only.

CONCLUSIONS

Strongyloidiasis is a relatively common disease in Durban; it is frequently associated with malnutrition and with other helminthic infestations. It is a general disease, insidious in its manifestations, and carries a mortality. It should be considered as potentially serious.

Adequate well-balanced diets are capable of producing a general improvement in severe cases of infestation, but no effect is observed on any of the other helminths which may also be present, and viable strongyloides larvae continue to be excreted in the faeces. A cure for strongyloidiasis cannot be expected from diet alone.

Dithiazanine (Partel) is an excellent therapeutic agent for strongyloidiasis; the action of this drug takes place in the gastro-intestinal tract. A partial success is claimed for thiocyanate therapy. Unlike dithiazanine, this drug has its effect in the body and not in the lumen of the gut, but the site of action of the drug was not determined. There was no effect noted on any other worm. Potassium thiocyanate is not as potent a vermicide as dithiazanine and is considerably more dangerous to use.

No reliance can be placed on eosinophil counts either as a guide during therapy or as a check for total eradication of this parasite. It is emphasized that the only satisfactory test of cure is repeated examinations for actual larvae.

SUMMARY

Strongyloidiasis is relatively common in Durban and deaths are known to occur from heavy infestation.

A small series of cases were treated by a variety of specific drugs. It was confirmed that dithiazanine (Partel) was the drug of choice, but other drugs may play a minor role in the elimination of this worm. A brief discussion is given on the value of thiocyanate therapy in this condition, and its difference from dithiazanine as regards site of action is commented on.

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FAMILIAL DYSAUTONOMIA

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'Son of man, behold, I take away from thee the desire of thine eyes . . . , neither shall thy tears run down.'
Ezekiel, ch. 24, v. 16.

'Mine eyes do fail with tears, my bowels are troubled, . . .'
Lamentations, ch. 2, v. 11.

On 21 September 1959 I examined a 4½-year-old boy who was losing weight and had severe emotional difficulties.

He was the first child of young healthy parents and was born in a nursing home after an uneventful full-term pregnancy. Because of premature rupture of the membranes and an impression of a large foetal head, Caesarian section was performed and a healthy male infant of 6 lb. 8 oz. delivered. Two years later a full-term brother of 6 lb. 3oz. was delivered *per vaginam*.

The patient was difficult to rear and was delayed in the acquisition of motor skills. He walked alone at about 2 years 4 months. He was an unhappy child, often restless and crying. At 20 months ulcerations on both corneae were cauterized. At 2½ years he was rather chubby, weighing about 30 lb., but he was inordinately restless and the possibility of retardation was mooted by a physician. As a consequence, he was taken to 'every doctor in town' and there was general agreement that he had suffered an undetected encephalitis in infancy which had left him with some retardation and with the emotional and hyperactive responses of the brain-damaged child. An attempt at I.Q. testing at 3 years suggested that he was 'about one year behind'. Various medications had little effect on his restlessness and excitability.

His best weight had been 34 lb. but in the last 6 months he had lost 4 lb. During these months he had been subject to episodes of diarrhoea and frequent upper respiratory infections.

Physical examination was virtually impossible. A small thin child, he would not lie down and would not stand still or sit for more than a few seconds. He darted hither and thither, restlessly energetic, compulsively touching everything new he saw—spatula, stethescope, ophthalmoscope and various objects round the room when he broke free. Yet he answered intelligently and to the point when questioned, though his own inquiries were more urgent and frequent. 'What's that?' he asked of everything new he saw. He saw with difficulty, for there were corneal opacities over both pupils, and he had to put his face close to an object in order to identify it, feeling it with both hands at the same time. The retinae were momentarily seen and seemed normal.

The skull appeared rather large for the boy's size, and was asymmetrical. There were no other physical signs considered to be of significance, and it was agreed that he presented the typical picture of the brain-injured child as commonly seen after an episode of encephalitis. If for no other reason than that his head appeared unduly large, a diagnosis of prenatal toxoplasmosis was entertained and an X-ray of the skull suggested. This showed no calcifications. Urinalysis and patch test were negative.

It seemed that the loss of weight was due to the recent upper respiratory infections. The use of benadryl was suggested for curbing his hyperkinetic behaviour. Despite his ceaseless activity and odd appearance and manner he nevertheless gave the impression that his intelligence was probably reasonably normal.

Some weeks later, far into the evening, my thoughts idly and unaccountably wandered to a day in 1952 when I had seen an unusual case on the wards of the Babies' Hospital in New York City, and in a flash it was clear that my patient had the same condition. Early the next morning two questions were put to the mother over the telephone, and the diagnosis was established:

'Is it correct to say that your little boy never produces tears?'

'That is so, doctor, he never has tears, no matter how much he cries. The eye doctor always asked me that question. Once he even tried to make him cry, but he still did not shed any tears. His eyes are moist, but tears are absent. In this respect he is quite different to my other child.'

'And is it true to state that he is somewhat insensitive to pain?'

'Pain? He doesn't know what pain is. It is amazing what he can endure. Last week he fell and dislodged four teeth: not a murmur. He is always falling and cutting and bruising himself, but somehow he doesn't seem to feel anything. I get the impression that he has been hurt and injured so often that he has become immune to pain. Mind you, he hates to have an injection, but the ordinary pains from injuries don't bother him a bit.'

The diagnosis was familial dysautonomia. Further inquiries revealed the following additional information:

Both parents are Jewish and unrelated, with no known familial traits. The mother became pregnant at an inconvenient time and half-hearted attempts were made to interrupt the pregnancy at 6 weeks by the use of an ergot preparation. The mother still feels guilty about this episode. Pregnancy proceeded normally and his birth by Caesarian section took place on 2 June 1955. Breast feeding was abandoned after a few days; he would not feed well, but the bottle was a poor substitute. His feeding was a great struggle; he often took more than an hour to swallow but a few ounces. When solids were added he was even worse. Throughout infancy he was fussy, unhappy, crying, refusing to feed despite evident hunger. It says much for the mother's perseverance that he weighed about 30 lb. at 2½ years.

The routine immunizations did not upset him unduly, and he has not yet suffered any of the 'inevitable' infections.

Teeth appeared at 7 months, he sat alone at 9 months, crawled at 14 months, attempted to walk with assistance at 18 months and walked alone at 2 years 4 months.

He was born with a squint in the left eye, which was corrected by operation at 2½ years. At 8 months ulcerations appeared on both corneae. These cleared and reappeared repeatedly, and at 20 months he was admitted to hospital, where the ulcerations were cauterized, after which the corneal scars did not progress, although they seriously interfered with his vision. At the time of his admission to hospital a number of investigations were performed as follows:

Hb. 12·7 g.%; w.b.c. 10,700 per c.mm. (polymorphs 62%, lymphocytes 32%); platelets normal; ESR 38 mm./hour. No pyrexia. Tuberculin test (PPD) negative. Numerous modifications of WR test, all negative. Serum albumin 2·8 g.%, globulin 2·7 g.%, gamma globulin 0·99 g.%. X-ray of hips (done because of odd gait) normal.

Home again, his behaviour was a sore trial. He had inexhaustible energy, never sat still, touched everything within sight, never finished a job, had no continuity of attention, and demanded an inordinate amount of parental affection and expressions of love. He could say 2 consecutive words ('I'm hot') at a year, but the mother feels that he did not really understand what he was saying until well after 2 years. At present he knows colours, can count up to 20, sings, recites nursery rhymes, is shrewd, thinks intelligently.

He eats well, usually sleeps well, has had no episodes of unexplained fever, no fits, is not prone to vomiting, but he drools excessively. His hearing and 'balance tubes' have been tested by an otologist and pronounced normal. His sense of smell is good. As an infant he was prone to long sessions of head banging, and he still indulges in head rolling, but was never given to thumb-sucking. There has been no difficulty with his sphincters. He walks with an unsteady gait. He is said not to sweat excessively, though 2 years ago he had an episode of fever associated with copious perspiration, and in the past month a nursery school teacher reported that his socks were wet with perspiration, and this on a cool day. His extremities have always been cold ('hands are like blocks of ice').

He was re-examined—again with some difficulty—and a number of additional features noted, as follows:

He is distinctly small (height 39 inches) and thin (his legs look like chicken-bones). The slightly asymmetrical skull measures 19¾ inches. A small patch of partial baldness is present in the right parietal region. The pulse is 90 per minute and regular. The blood pressure is 100/70 mm. Hg in the supine position, and falls to 96/64 mm. on standing up, but promptly jumps 10 mm. (systolic and diastolic) when anything distracts or excites him—which is pretty often. His eyes are pale blue and have corneal opacities over both pupils, the right opacity being rather larger and denser. The corneae are totally anaesthetic; he fails to blink no matter how long cottonwool is rubbed over them. There were 2 or 3 healed scars on his thin bird-like face. His voice lacked the crisp clear tones of childhood. Despite the warmth of the day his hands were remarkably cold and the skin on the arms and legs was blue and mottled. Peripheral pulses normal. There was no scoliosis, no pes cavus, no dermographism. Knee jerks were brisk and equal. The gait was unsteady but he had no spasticity or undue flaccidity. Gag reflex present. He was drooling.

In February 1959 an EEG was done while he had been put to sleep with barbiturates: 'There are a large number of scattered high-amplitude delta waves. This is probably due to moderate brain damage from an early age; in fact many of the waves resemble space-occupying lesion waves, non-focal and non-episodic, though there is some predominance in the left occipital area' (Dr. L. Sussman, Tara Hospital).

The X-ray of the skull which was done to exclude toxoplasmosis, though otherwise normal, indicated the presence of plagiocephaly. There was no suggestion of increased intracranial pressure. The parents declined to have air studies performed.

DISCUSSION

This patient has a hotch-potch of curious and seemingly unrelated symptoms and signs, and one may readily be excused for overlooking the fact that a symptom-complex exists. The most pronounced features of his syndrome are: Jewish origin, lack of tears with corneal anaesthesia and opacities, insensitivity to pain, poor motor coordination, emotional lability, difficulty with feeding in infancy, cold extremities.

These features apparently have no connection with each other, but Riley and his co-workers[1] (1949) recognized the presence of a specific entity, described the syndrome and gave it a name.

At the Babies' Hospital, New York City, they observed 4 children 'with symptoms so puzzling as to defy exact diagnosis yet so similar as to constitute a clinical entity'.[1] Since then (up to 1957) about 70 cases of the Riley-Day syndrome have been identified in North America, and one reported from Germany and one from England.[2,3]

Riley states[2] that the manifold symptomatology is clearly due to widespread involvement of the central nervous system—ranging from autonomic, through motor and sensory, to psychic disturbances, and that such widespread derangements can only be conceived of in terms of biochemical malfunction—possibly some enzymatic defect of metabolism within the central nervous system. He lists the signs and symptoms under 3 headings:

(*a*) *Probably present in all cases, though future experience may reveal exceptions:* Jewish origin (with very rare exceptions); reduced tears when crying; emotional lability; indifference to pain; postural hypotension; excessive perspiration; cold hands and feet; disturbed swallowing reflex; poor motor incoordination; dysarthria.

(*b*) *Often present:* Hypertension on excitement; skin blotching on excitement; erratic temperature control; drooling beyond the usual age; hypoactive or absent tendon jerks; corneal anaesthesia; mental retardation (in about half of all cases); breath-holding spells in infancy; episodes of vomiting; frequent bronchopneumonia; retardation in growth; abnormal EEG; occurrence in siblings (25% - 35% of cases).

(*c*) *Sometimes present:* Diarrhoea or constipation; scoliosis and other orthopaedic conditions; corneal ulceration; increased tendency to body injury; consanguinity of parents; occurrence in related families.

Children with dysautonomia that are prone to recurrent vomiting, bronchopneumonia and hyperpyrexia have a poor prognosis, and 13 of the described cases have come to autopsy, but have shown no consistent gross abnormalities of the central nervous system.[2] Air studies of the brain are generally unhelpful[4] and the blood chemistry normal,[5] but the EEG is non-specifically abnormal.[4] The diagnosis is thus entirely clinical.

The disease is virtually confined to Jews, though Riley believes that it has been satisfactorily defined in 4 non-Jewish children.[2] This might perhaps be due to unknown Jewish antecedents, genetic mutation, or prenatal phenocopy. Jews have their troubles, not the least of them being their genetic disabilities, and, since they intermarry, dysautonomia (recessive gene?) virtually remains confined to them. Its alleged presence in non-Jewish children should be viewed with much suspicion. In about a quarter to a third of the reported cases siblings have been affected.

The principal diagnostic feature is the lack of tears, which together with corneal anaesthesia is responsible for the occurrance of corneal ulceration and cicatrization. Biopsy of the lacrymal glands shows no abnormality.

All the children are dwarfed, and nearly all poorly nourished, with comparable delay in bone maturation, but puberty is not strikingly delayed. Their emotional and physical handicaps are responsible for the difficulty in accurately assessing their intellectual capacities, and their disturbances of behaviour are frequently the chief cause of concern within the family. They are slow to acquire motor skills and most begin talking late, and have a nasal and monotonous voice.

Frequently they develop transient erythematous blotches, usually about the upper chest, and they invariably have profuse perspiration (but not in the case reported here). The electrolyte concentration of sweat is normal. Unlike what happens normally, in all cases reported the blood pressure (especially the diastolic) drops when the patient

stands up (in the case reported here this was not a striking feature). Sometimes there are massive swings in blood pressure, leading to suspicion of phaeochromocytoma. Knee jerks may be present, especially in older children. Routine immunization procedures sometimes produce major feverish reactions, and unexplained hyperpyrexia is common; so are frequent bouts of bronchopneumonia. At about 3 - 4 years of age recurrent episodes of severe and prolonged vomiting may make their appearance, frequently requiring the use of intravenous fluids.

Differential Diagnosis

According to which constellation of symptoms is most prominent, it may be necessary to exclude such conditions as fibrocystic disease, pink disease, hypogammaglobulinaemia, mental retardation, childhood schizophrenia, cerebral palsy, Sjorgen's syndrome, and phaeochromocytoma. In the full-blown case of dysautonomia the diagnosis presents but little difficulty.

Prognosis and Treatment

There is insufficient data for advice on prognosis, but certainly the death rate is higher in those prone to infections, hyperpyrexia and vomiting. The oldest recorded patient is now (1959) 25 years old. Up to 1957 the condition had not been described *de novo* in adults. The patients' emotional difficulties tend to subside with time and, if their intellect is reasonable, their adaptation to society may be adequate.

The risks inherent in anaesthesia have not yet been properly assessed; more data are needed. My patient had two uneventful operations on his eyes.

Treatment is entirely supportive. The child here reported is making progress in a nursery school for emotionally disturbed children. The psychiatric aspects of the condition have been considered by Freedman *et al.*[5] A society for the parents of afflicted children has been formed in New York and group therapy has proved helpful.

SUMMARY

The Riley-Day syndrome or familial dysautonomia has not yet been reported in this country. A case of this 'new disease' is now recorded in a 4-year-old Jewish boy, who illustrates the principal features of lack of tears with corneal anaesthesia and corneal ulcers, insensitivity to pain, cold extremities, pronounced feeding difficulties, motor incoordination, dysarthria and emotional instability—in particular, anxiety, restlessness, impulsiveness, irascibility, compulsive touching, and hyperkinesia, in fact the typical picture of the behavioural disturbances of the brain-injured child. The absence of marked postural hypotension and of constant excessive perspiration is not felt to be a bar to the diagnosis.

The diagnosis is entirely clinical, and no doubt *formes frustes* of the condition occur—all the way to normality. Until more definitive diagnostic criteria are established (e.g. one or other enzyme, yet to be isolated, in the plasma or cerebrospinal fluid), it would be difficult indeed to sustain a diagnosis of 'a mild case of dysautonomia'. The syndrome lends an interesting foundation to the possibility of organic brain damage (enzymatic defect?) as a basis of some disturbances of behaviour.

There is no treatment other than supportive and there are no consistent gross brain abnormalities to account for this conglomeration of curious symptoms. The syndrome is familial, and virtually confined to Jews.

My thanks are due to Dr. K. F. Mills, Superintendent of the Johannesburg General Hospital, for permission to quote from the patient's records at the Children's Hospital, and to Dr. H. Moross for allowing me to reproduce the EEG report from Tara Hospital.

POSTSCRIPT

I have since been informed by Dr. B. Senior, Johannesburg, that his firm of paediatricians have seen two or three children with dysautonomia in the past few years. The case reported here was also seen by one of them, and I am given to understand that the diagnosis of dysautonomia was entertained but was not communicated to the parents.

Since this report was submitted the mother has been delivered of another boy; he is breast feeding normally and is presumed to be well.

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3. McKendrick, T. (1958): Arch. Dis. Childh., **33**, 465.
4. Riley, C. M. (1952): J. Amer. Med. Assoc., **149**, 1532.
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ASSOCIATION NEWS : VERENIGINGSNUUS

RAILWAY MEDICAL OFFICERS' GROUP

On the occasion of the retirement of Dr. L. O. Vercueil, after 30 years service as a railway medical officer, as well as Chairman of the RMO Group, his colleagues of the RMO Group, bid him farewell at a most enjoyable cocktail party held in Johannesburg on the evening of 6 April.

The party was well attended and many railway medical officers from Johannesburg and the Reef were present. Also present, amongst other representatives from the Railways Administration and the Sick Fund, were Mr. D. H. C. du Plessis, General Manager, South African Railways, and Mr. J. P. Hugo, Deputy General Manager of the SAR.

Dr. H. Penn, Vice-Chairman of the RMO Group, who presided, paid a glowing tribute to the sterling services Dr. Vercueil had rendered to the RMO Group during his 10 years of office as Chairman of the Group, and on behalf of the RMO Group made a presentation to Dr. Vercueil of a beautifully executed illuminated address, as well as a radio and cheque.

Both Mr. du Plessis and Mr. Hugo spoke of the great help Dr. Vercueil had been at all times to the Sick Fund, and of the prominent part he played in the introduction, several years previously, of the 'limited choice of doctor' scheme. Mr. du Plessis also made a presentation to Dr. Vercueil on behalf of the SAR Sick Fund.

Drs. E. W. Turton and M. Cohen also paid tribute to the great services Dr. Vercueil had rendered to the RMO Group and expressed great regret that his dynamic leadership will now be lost to the Group. Dr. Vercueil replied suitably.

CAPE WESTERN BRANCH

On 29 February the Cape Western Branch of the Medical Association held a Grand Première of the film *Quiet Flows the Don* at the Odeon Theatre, Sea Point, in aid of the Benevolent Fund of the Association. The Première was preceded by a braaivleis and hat parade and it is generally agreed that the evening was most successful. Dr. H. O. Hofmeyr, the convener, and his committee are to be congratulated on raising an amount of £692 for the Benevolent Fund.

CENTRAL COUNCIL FOR HEALTH EDUCATION

1960 SUMMER SCHOOL IN HEALTH EDUCATION

'Making health matter' is the title of the 1960 Summer School in Health Education, which will take place from 9 to 19 August at the Froebel Educational Institute in London. The greatest part of the programme is arranged on a new plan, which omits the formal group discussion periods of previous years. In their stead several 'forums' have been devised at which experts in the subjects under consideration will present short papers as a basis for the discussion of such topics as juvenile delinquency, the community care of the mentally ill, recent progress in medicine, and the problems of public health workers. One experimental session for group discussion is planned, at which an attempt will be made to measure the effectiveness of group discussion in attitude changing. More time has been allowed for practical work, and the lecture-demonstrations on the techniques of health education have been retained.

A wide range of health education material selected from various countries will be on view and modern projection and recording equipment will be available for demonstration and teaching purposes.

Delegates will be allocated individual study bedrooms. The inclusive fee for the course will be £22 0s. 0d. (Tuition fee £9 10s. 0d., residence £12 10s. 0d.) On registration 2 guineas of the total fee is payable, and this is not returnable after 9 July except in cases of personal illness when a medical certificate will be required.

Application to attend should be made to the Medical Director, The Central Council for Health Education, Tavistock House, Tavistock Square, London, W.C. 1.

OFFICIAL ANNOUNCEMENT : AMPTELIKE AANKONDIGING

AMENDMENT OF BY-LAWS

Notice is hereby given that at the Meeting of Federal Council held in Pretoria in March 1960, the following amendments to the By-laws of the Association were finally approved in terms of By-law 69:

(i) That *By-law* 9 (*c*) be amended to read: 'In the case of husbands and wives residing together, both being members of the Association, the subscription payable by the wife shall be two guineas less than the normal subscription to the Association'.

(ii) That in *By-law* 30 the words 'in person or by proxy' be inserted after the words 'Except as hereinafter provided in this By-law, no business shall be transacted in any General Meeting unless there be present a quorum of not less than fifty members'.

(iii) That *By-law* 41 be amended so that the words 'The Council shall meet not less than twice a year and shall be presided over by the Chairman of Council' be altered to read: 'The Council shall meet at least once a year and shall be presided over by the Chairman of Council'; and that the following words at present appearing in the By-law be deleted: 'The Chairman shall have power, however, to decide that any meetings shall not be held if, in his opinion, there is not sufficient business of importance, and due notice of the postponement shall be sent to all members of the Council. It shall not, however, be competent to be postponed for two consecutive meetings'.

(iv) That the first paragraph of *By-law* 58 be amended to read: 'There shall be elected at the first meeting of the Council following each triennial election a Head Office and Journal Committee which shall consist of the President, the Chairman, the Vice-Chairman, the Honorary Treasurer and five members of Council, with power to coopt'.

By Order of the Council

A. H. Tonkin
Secretary

Medical House
Cape Town
22 April 1960

WYSIGING VAN VERORDENINGE

Kennis word hiermee gegee dat die volgende wysiginge van die Verordeninge van die Vereniging finaal goedgekeur is in terme van Verordening 69, op die vergadering van die Federale Raad wat in Maart 1960 in Pretoria gehou is:

(i) Dat *Verordening* 9 (*c*) gewysig word om te lees: ‚In die geval waar 'n man en sy vrou saamwoon en albei lede van die Vereniging is, sal die subskripsie betaalbaar deur die vrou twee ghienies minder wees as die normale subskripsie van die Vereniging'.

(ii) Dat in *Verordening* 30 die woorde ‚persoonlik of per volmag' ingevoeg word na die woorde ‚Behoudens enige bepalinge in hierdie Verordening hierna, mag geen sake op enige algemene vergadering afgehandel word tensy daar 'n kworum van minstens vyftig lede teenwoordig is nie'.

(iii) Dat *Verordening* 41 gewysig word sodat die woorde ‚Die Raad vergader minstens twee maal per jaar, onder voorsitterskap van die voorsitter van die Raad', soos volg sal lees: ‚Die Raad sal minstens een keer per jaar vergader onder voorsitterskap van die voorsitter van die Raad'; en dat die volgende woorde wat nou in die Verordening voorkom, uitgelaat word: Die voorsitter is egter by magte om te besluit dat enige vergadering nie gehou sal word nie as daar volgens sy mening nie genoeg sake van belang is om behandel te word nie; behoorlike kennisgewing van die verskuiwing moet aan alle lede van die Raad gestuur word. Twee opeenvolgende vergaderings mag egter nie verskuif word nie'.

(iv) Dat die eerste paragraaf van *Verordening* 58 gewysig word om te lees: ‚Op die eerste vergadering van die Raad na elke driejaarlikse verkiesing moet 'n Hoofkantoor- en Tydskrifkomitee gekies word wat moet bestaan uit die President, die Voorsitter, die Vise-Voorsitter, die Ere-penningsmeester en vyf lede van die Raad met die mag om te koöpteer'.

Op Las van die Raad

A. H. Tonkin
Sekretaris

Mediese Huis
Kaapstad
22 April 1960

NAWEEKKURSUS IN PSIGIATRIE VIR ALGEMENE PRAKTISYNS

Dit is besluit om algemene praktisyns uit te nooi na 'n naweekkursus in ‚Psigiatrie vir die algemene praktisyn' te Tara-hospitaal vanaf 20 tot 22 Mei 1960. Die Kursus is beplan om van belang vir die algemene praktisyn te wees en word gehou in samewerking met die Vereniging van Mediese Gegradueerdes van die Universiteit van die Witwatersrand. Die registrasiegelde is £3. 3s. 0d. en die opbrings sal na die Hospitaalbiblioteek van Tara gaan.

PROGRAM

Vrydag 20 Mei

6.00 nm. Verwelkoming en sjerrie.
7.00 nm. Eetmaal.
8.00 nm. Openingstoespraak: Prof. L. A. Hurst ‚Psigiatrie in die algemene geneeskunde' en dr. H. Moross ‚Die plek van psigiatrie in gesondheidsdienste.'

Saterdag 21 Mei

9.00 vm. Fisiese metodes van behandeling. Dr. M. Feldman.
10.15 vm. Tee.
10.30 vm. Psigiatriese noodgevalle in algemene geneeskunde. Dr. A. Sidley.
12.00 nm. Voorstelling van gevalle. Drs. D. Perk, M. Feldman, B. Levinson, en B. Braude.
1.00 nm. Middagete.
2.00 nm. Psigiatriese behandeling. (*a*) Psigofarmakoterapie. Prof. L. A. Hurst. (*b*) Voorstelling oor psigoterapie. Drs. M. Klass en L. S. Gillis.
3.30 nm. Tee.
4.00 nm. Angs by kinders. Dr. F. Reinhold.

Sondag 22 Mei

9.00 vm. Gewone psigosomatiese toestande. Prof. G. A. Elliott.
10.15 vm. Tee.
10.30 vm. Psigologiese probleme in die huwelik. Dr. F. Frankel.
12.00 nm. Rolprent oor alkoholisme gevolg deur bespreking. Drs. H. E. van Hoepen en M. C. Frame.
1.00 nm. Middagete.
2.00 nm. Psigiatriese probleme by die bejaardes. Dr. D. Perk.
3.30 nm. Tee.
4.00 nm. Neurologiese probleme by die bejaardes. Dr. S. M. Katz.

Aansoeke om die kursus by te woon moet so vroeg moontlik gerig word aan die Mediese Superintendent, Tara-hospitaal, Posbus 13, Saxonwold, Johannesburg.

PASSING EVENTS : IN DIE VERBYGAAN

Register of Medical Practitioners and Dentists. The register of medical practitioners and dentists, registered as at 1 January 1960, has now been published, and is available for sale to the profession and the public. The cost of the register is £1 5s. 0d. for an ordinary copy, and £1 10s. 0d. for an interleaved copy. In addition, monthly amendments to the register may be obtained on the payment of 12s. for the year. The register is obtainable from the Registrar, South African Medical and Dental Council, P.O. Box 205, Pretoria.

Register van Geneeshere en Tandartse. Die register van geneeshere en tandartse wat op die register was op 1 Januarie 1960, is nou gepubliseer en dit kan deur die publiek gekoop word. Die prys van die register is £1 5s. 0d. vir die gewone deel en £1 10s. 0d. vir 'n deel met tussenblaaie. Maandelikse wysiginge van die register kan ook verkry word teen 'n betaling van 12s. vir die jaar. Die register is verkrygbaar van die Registrateur, Suid-Afrikaanse Geneeskundige en Tandheelkundige Raad, Posbus 205, Pretoria.

* * *

Mr. Ben Bellon, ear, nose and throat surgeon, has left Johannesburg for the Continent and England, where he will visit otological clinics. He will be away for 2½ months and will return to South Africa on 14 July 1960.

Dr. Ben Bellon, oor-, neus- en keelarts, het van Johannesburg vertrek na die Vasteland en Engeland, waar hy otologiese klinieke sal besoek. Hy sal 2½ maande lank weg wees en sal op 14 Julie 1960 na Suid-Afrika terugkeer.

* * *

University of Cape Town and Association of Surgeons of South Africa (M.A.S.A.) Joint Lectures. The next lecture in this series will be held on Wednesday 11 May at 5.30 p.m. in the E-floor Lecture Theatre, Groote Schuur Hospital, Observatory, Cape. Mr. T. L. Sarkin will speak on 'Tuberculosis of bone'. All members of the Medical Association are welcome.

On Thursday 12 May Mr. Douglas Robb, Sims Travelling Professor for 1960, of Auckland, New Zealand will lecture on 'Arterial occlusive disease—aorto-iliac, femoral and carotid' at 5.00 p.m. in the Falconer (E-floor) Lecture Theatre, Groote Schuur Hospital.

* * *

South African Institute for Medical Research, Johannesburg, Staff Scientific Meeting. The next meeting will be held on Monday 16 May at 5.10 p.m. in the Institute Lecture Theatre. Dr. N. S. F. Proctor will speak on 'The United States Armed Forces Institute of Pathology and some of its contributions to pathology and neurology'.

* * *

Members are reminded that they should notify any change of address to the Secretary of the Medical Association of South Africa at P.O. Box 643, Cape Town, as well as to the Registrar of the South African Medical and Dental Council, P.O. Box 205, Pretoria. Failure to advise the Association will result in non-delivery of the *Journal*. This applies to members proceeding overseas as well as those who change their addresses within the Union.

* * *

Research Forum, University of Cape Town. The next meeting of Research Forum will be held on Wednesday 18 May at 12 noon in the Bennie de Wet Lecture Theatre, A-floor, Groote Schuur Hospital, Observatory, Cape. Dr. H. E. Schendel will speak on '(1) Partition of urinary nitrogen during treatment of protein-depleted infants, and (2) A comparative study of biochemical indices used in evaluating dietary protein' (by Drs. H. E. Schendel and J. D. L. Hansen). All who are interested are invited to attend this meeting.

* * *

Dr. David Glajchen, M.B., B.Ch. (Rand), M.R.C.P. (Edin.), physician, has commenced practice at 710 Medical Centre, Jeppe Street, Johannesburg. Telephone: Consulting room 22-9440, emergency 22-4191. These numbers do not appear in the current Telephone Directory.

* * *

Dr. J. D. L. Hansen, of the Department of Child Health, University of Cape Town, has been awarded a Rockefeller Travel Grant to visit nutrition and paediatric centres in the USA, Central America and the UK.

* * *

Mr. P. J. Mitford Retief, Ch.M., accompanied by his wife, will leave Cape Town on 20 May to attend a meeting of the British Association of Urological Surgeons in London on 22 - 26 June. Mr. Retief hopes to visit the main urological centres in the UK and will be away for 2 months.

* * *

Dr. Roy Morris, M.D., M.R.C.P.E., left Johannesburg on 4 May for a 2-months' visit to various medical clinics in the USA and Great Britain.

* * *

Advertisement for Contract Appointments. The attention of members of the Association is drawn to the possibility of advertisements for contract appointments appearing in journals other than the *South African Medical Journal*. When an advertisement for an unapproved appointment is published in the *South African Medical Journal* a notice is placed at the same time advising practitioners to communicate first with the Hon. Secretary of the Branch concerned before applying for the appointment. When advertisements are published in other journals, there is no indication of the acceptability or otherwise of the appointments advertised. It would therefore be in the interest of practitioners for them to ascertain from the local Branch of the Association whether an appointment advertised in another journal carries the approval of the Association or not.

* * *

Tak Griekwaland-Wes (M.V.S.A.). Tydens die onlangse Jaarvergadering van die Tak, is die volgende Takraadslede gekies: Voorsitter: Dr. H. Lowenthal: Ondervoorsitter: Dr. D. Keys; Sekretaris: Dr. J. D. Visser; Ondersekretaris: Dr. R. Amoils; Eretesourier: Dr. N. Kretzmar; addisionele lede: Drs. A. B. de Villiers Minnaar, J. Kieser, H. Beyleveld, S. Perel and G. Jacobs.

BOOK REVIEWS : BOEKBESPREKINGS

NEUROLOGY FOR MEDICAL STUDENTS

Aids to Neurology. 2nd edition. By E. A. B. Pritchard, M.A., M.D., F.R.C.F. Pp. x + 480. Illustrations. 15s. London: Baillière, Tindall and Cox Ltd. 1959.

Like all the volumes in the 'Aids' series, this book is written primarily for the medical student. It seeks to provide the basic essentials of neurology for his clinical years of study. The various neurological syndromes and diseases are described in a summarized form suitable for easy reference. Physical signs elicited by examination are generally described in terms of the tests carried out and the observations made.

The book is divided into 5 sections and deals with the clinical disorders of the peripheral nervous system, of the spinal cord, of

the brain, and of the vegetative nervous system. The final section is devoted to an outline of neuro-anatomy and neurophysiology, showing how these subjects throw light on the clinical material described and the interrelation of the various clinical symptoms. It also includes a scheme for routine examination of the nervous system.

Undoubtedly a useful 'aid' to the student.

A.H.T.

VENTILASIE VAN DIE LONGE

Automatic Ventilation of the Lungs. Deur William W. Mushin, M.A., M.B., B.S., F.F.A.R.C.S., F.F.A.R.A.C.S., L. Rendell-Baker, M.B., B.S., F.F.A.R.C.S. en Peter W. Thompson, B.A., M.B., B.Chir., F.F.A.R.C.S. Hoofstukke oor *Physics of Automatic Ventilators*, deur W. W. Mapleson, Ph.D., A.Inst.P. Pp. xvi + 349. 191 afbeeldings. 47s. 6d. Oxford: Blackwell Scientific Publications. 1959.

Die gebruik van spierverslappers as 'n roetine en die tegniek van doelbewuste apnee met gekontroleerde ventilasie het in die afgelope 20 jaar grootskaalse ontwikkeling meegebring op die gebied van meganiese ventilators. Die aansienlike verskeidenheid ventilators met gekompliseerde wisseling van fisisiese en meganiese beginsels waarvolgens hulle fungeer, het vir die gemiddelde narkotiseur bykans 'n geestesverwarring geskep.

In hierdie lofwaardige handboek slaag die skrywers daarin om hierdie ingewikkelde aspek van moderne narkosetegniek klinkklaar te verduidelik. Binne die bestek van 2 hoofstukke word 'n oorsig gebied van die fisiologiese en kliniese faktore wat gepaard gaan met die tegniek van gekontroleerde ventilasie.

Ter inleiding tot die beskrywing van 60 verskillende ventilators bied Dr. W. W. Mapleson 2 hoofstukke waarin die fisiese grondbeginsels van hierdie masjiene bespreek en geklassifiseer word. Ons beskeie mening is dat hierdie afdeling van die handboek die gemiddelde leser sal afskrik. Uit die pen van 'n geskoolde fisikus spruit hier pitkos wat bokant menige praktiserende narkotiseur se vuurmaakplek sal wees. In 'n werk wat spesifiek aan praktiserende narkotiseurs gerig word, waarsku ons teen die gevaar dat bydraes van verwante wetenskappe ongelukkig van so 'n gespesialiseerde aard mag wees dat dit die leser die kluts laat kwytraak.

Die skematiese voorstellings en beskrywings van die ventilators is onoortreflbaar. Dit tref veral weens die saaklike eenvoud wat vir die leser wat onvertroud mag wees met besondere masjiene onmiddellik duidelikheid meebring.

Hiermee 'n handboek van onskatbare waarde vir alle narkotiseurs, asook vir interniste wat o.a. tydens die behandeling van poliomiëlitis, tetanus, miastenie gravis, en vergiftiging met verdowingsmiddels voor respiratoriese probleme te staan kom.

J.A.P.

GROUP PSYCHIATRY

Group Psychotherapy — Theory and Practice. 2nd edition. By J. W. Klapman, M.D. Pp. x + 301. $6.75. New York and London: Grune & Stratton, Inc. 1959.

Revising a 13-year-old book on group psychotherapy, written when this treatment procedure was in its infancy, imposes a difficult task on its author. Only recently, as controversial issues have been clarified, has a coherent treatment discipline begun to emerge.

Some consider the origin of this potent means of psychologieal treatment to lie in 1905, when J. H. Pratt organized his patients with tuberculosis into groups, for imparting to them rules of medical hygiene; in addition to medical instruction, they obtained from their group membership definite encouragement and renewed self-esteem. However, these unsophisticated classes were not psychotherapeutic in considered design. Moreno is usually credited with founding group psychotherapy, in Vienna during the first world war. After the last war, in the 'forties, group psychotherapy had established itself by proving effective in treating combat neurosis.

A text-book that first appeared in 1946 had therefore already for subject a wide field of experience and a diversity of clinical approaches; there was justification for describing a confusing range of often ill-defined, ambiguous, uncritically-applied treatment practices. Dr. Klapman has not used the opportunity offered by a new edition to eliminate obsolete theorizings in favour of recent clarifications. An alert doctor wanting to inform himself of what group psychotherapy is supposed to do, what theory it rests upon at present, what actual clinical results it obtains, will probably set this book aside dazed and sceptical. The reader cannot but be confused when group psychotherapy is held to include everything from intensive, closed, long-term groups up to such oddities as library-book discussions. The author does not exert himself critically to arrive at definition in precise terms of what constitutes the rationale of group psychotherapy. Nebulous theorizing predominates. The technique which the author himself uses and recommends does not emerge plainly. The chapter on 'Assessment and results of group psychotherapy' is meagre and dodges the clinical issues.

The medical interest evoked by group psychotherapy is too considerable to permit this volume to pass as representative of contemporary serious writing on the subject. This treatment method merits more scientific appraisal than Dr. Klapman has accorded it, for weighty claims are being made for it. Group psychotherapy has been held to be a treatment producing a lasting change in personality, an invaluable method for teaching psychotherapeutic principles to doctors, and a tool for research in the social sciences. To convey the impression that merely collecting people into a group implies that therapy will occur is to undermine what advances have been made in this field.

H.W.

CORRESPONDENCE : BRIEWERUBRIEK

THE NEW APPROACH TO MEDICAL AID SOCIETIES

To the Editor: I wish to congratulate you on your Editorial[1] in the issue of the *Journal* for 9 April 1960, under the heading 'The new approach to medical aid societies'. It has certainly cleared the air as far as I am concerned except for a few pertinent questions which I should like to ask.

1. Are all patients of these insurance companies to be treated as private patients and charged at normal private rates until certain groups insured by them receive recognition and qualify for the preferential tariff?

2. What is my position when the patient proffers me the cheque received by the insurance company in part payment of my account and indicates to me that I am expected to accept this in full payment?

3. What is to be the position of people earning over £2,500 under the new groups—also those who are now below £2,500 but who will eventually exceed this mark. Your Editorial does not appear to agree with a questionnaire from an insurance company which gives the impression that all members, whether they earn £2.500 or more, will get preferential tariff provided the group scheme complies with the conditions laid down by the Medical Association. (*Vide* SANSOM'S letter dated 22 March 1960.)

4. Can the Association be guaranteed that accurate information will be obtained on these groups in view of the difficulty of divulging earnings?

5. What will be the position when the patient is in arrears with the 20% he owes to the company. Is it true that the doctor will not be paid? (See report of Managing Director of SANSOM at their Annual General Meeting in March.)

6. Would it not be wise if Federal Council, through your columns, kept the profession informed of all developments, as it appears that the antagonism against the decisions of Federal Council regarding insurance companies derive from either lack of information or false information?

7. The last point is of great importance owing to the tactical advantage gained by one of the insurance companies by issuing statements long before the cumbersome machinery of Federal Council could be geared to make a statement to its members.

L. Wunsh

605 Harley Chambers
Jeppe Street
Johannesburg
21 April 1960

1. Editorial (1960): S. Afr. Med. J., 34, 295.

[The answers to your correspondent's questions are as follows:
1. Yes, private practice fees should be charged in accordance with the patient's normal financial standing.

2. A doctor is not bound to accept the cheque in full payment unless he is satisfied that it is an equitable amount for the service rendered and is reasonable in view of the patient's financial position.
3. Persons earning over £2,500 are not acceptable in 'groups' which may be recognized as medical aid societies. They may remain insured as private persons outside the groups. The same must apply to persons who eventually may earn over £2,500 as long as that figure is the 'ceiling'. The impression given in a circular letter, to which your correspondent has referred, is incorrect and the company has been informed of this.
4. At present we accept certificates from the secretaries of medical aid societies in good faith and we will no doubt accept the same from the companies and firms (groups) who submit them in support of an application for approval made through an insurance company.
5. Our agreement with the insurance companies lays down 'payment direct and in full to the doctor' and we cannot be concerned with loans owed to an insurance company by its members. The insurance company concerned has been informed of this.
6. The columns of the *Journal* will be used for this purpose and members will be wise to believe what they read officially in the *Journal* rather than what they may read or be told elsewhere.
7. The statement which appeared in the *Journal* of 19 March was prepared before information was given to the press, but in spite of this the daily papers have an advantage over a weekly publication.—*Secretary (M.A.S.A.)*]

NEW DISEASE IN UGANDA

The Secretary for Health of the Union of South Africa has kindly forwarded to the *Journal* a copy of a letter addressed by the Commissioner for the Union of South Africa in Nairobi to the Secretary of External Affairs. This letter is reproduced below:

'According to a press release published by the Department of Information, Kampala, Uganda, on 5 February 1960, the virus of a new disease which has affected half a million people in Uganda has been isolated by a team of specialists from the East African Virus Research Institute, Entebbe, and the Uganda Medical Department. The disease has swept across the Protectorate starting in West Nile and travelling through Northern Province and Eastern Province into Kenya.

The following information is passed on to you for submission to the Ministry of Health:

"Working more than 12 hours a day, sometimes in their laboratories at Entebbe, sometimes in the epidemic areas, the team isolated the new virus from the blood of patients and subjected it to tests in the brains of mice to check its identity. They found that it is closely related to Chikungunya virus which was isolated by the staff of the institute during a huge epidemic with much the same symptoms which broke out in Tanganyika 6 years ago.

"The new disease is characterized by headache, fever, swollen glands and crippling joint pains. Often it is followed after 4 days by an itching rash. These symptoms last for 5 - 7 days and are usually followed by complete recovery. No deaths have so far been reported.

"The disease was first investigated by the Acting Provincial Medical Officer in Northern Province, Dr. H. Shore, early in 1959. It advanced on a 100-mile broad front through Acholi, Lango and Teso and reached the Kenya border in November.

"The first task of the investigating team, which included Dr. M. C. Williams, Dr. P. S. Corbet, Dr. J. P. Woodall and Mr. J. M. Ellice of the Virus Research Institute and Mr. V. T. Odoi of the Uganda Medical Department, was to find out how the disease was caused and then how it was carried. The Uganda Medical Department made an immediate money grant to allow field work to be undertaken.

"The entomologists found that the disease was being carried by two species of anopheles mosquitoes, although up to then malaria-carrying mosquitoes had never been incriminated as transmitting virus epidemics anywhere in the world. To date the team has isolated 10 strains of the new virus from these two species of mosquito which are the principal malaria carriers in most parts of Africa.

"The new disease, known as *O'nyong-nyong*, i.e. *joint-breaker*, by sufferers because of its effects, has been largely confined to Africans because Asians and Europeans usually take precautions with nets and screens against being bitten by the night-biting malarial mosquitoes.

"The assistant Director of the Virus Research Institute, Dr. J. D. Gillett, said that the disease involved not only an apparently new virus, but also involved mosquitoes previously considered to be unimportant in the transmission of virus disease. It was extremely unpleasant while it lasted and because of its crippling nature had prevented many sufferers from getting to hospitals".

N. J. J. Jooste
Commissioner: Union of South Africa

Nairobi
20 February 1960'

THE DECLARATION OF GENEVA 1948

To the Editor: May I, through the medium of the *South African Medical Journal*, congratulate the Editor of the April 'Newsletter' of the Cape Western Branch (M.A.S.A.) on the editorial, and for drawing the attention of the Branch members to the Declaration of Geneva 1948.

The pledges of this Declaration—the revised version of the Hippocratic Oath—made at the time of being admitted as members of the medical profession, are not mere pious platitudes of a bygone age. They represent the considered world-medical opinion of *today*, as expressed by the 85-nation organization—the World Medical Association.

One paragraph of the leading article referred to is particularly worthy of note; viz. 'It behoves us all to remember this line from the International Code of Ethics: "A Doctor must not allow himself to be influenced merely by motives of profit".' This seems to be the direct antithesis of a suggestion made by a member of the Association at a recent meeting, that whole-time teachers of medicine should be paid £10,000 a year, or else be allowed private practice to increase their present salaries. This proposal also lacks a sense of proportion in that it disregards any consideration of the relative value to a university of whole-time teachers of medicine as compared with whole-time teachers in other faculties. Lecturers in other faculties have similar financial and social obligations to their medical colleagues, but apparently are expected to carry on their work at approximately one-fifth of the salary of medical lecturers. In the light of the implications of this proposal, is it to be wondered at that, on two occasions in the short space of 30 years, the Government of the day has taken steps to ensure that the public is not being exploited by excessive medical fees?

It has also been suggested that, unless whole-time clinical teachers are allowed to undertake private practice to supplement their incomes, the best men are unlikely to apply for such university posts. This outlook may be true in commerce, but should not apply to a vocation or calling and, in my opinion, is a reflection on the reputation of the profession of medicine.

During a recent visit to Great Britain, after discussions with a number of whole-time clinical teachers whose contracts prohibited private practice, I gained the impression that, whereas higher remuneration from the universities was desirable, these men have accepted the relatively small salaries out of a sense of obligation to the Declaration of Geneva, and the pledge 'to consecrate my life to the service of humanity', and to 'maintain, by all the means in my power, the honour and the noble traditions of the medical profession'.

A special duty devolves upon teachers of medicine, for their example and precept must necessarily influence their students, and it depends on them whether the ethical standards of medical practitioners are high or not.

It is to be hoped that, when appointing whole-time clinical teachers, the South African universities will bear in mind that it is possible to attach too much importance to academic qualifications at the expense of other qualities equally desirable in candidates; also, that it much depends on their choice whether future generations of practitioners look upon their occupation as a profession or a business.

The following are extracts from the Leading Article entitled 'Behaviour of the market place', published in the *British Medical Journal* of 7 November 1959:

'Our new President, Prince Philip, reminded us . . . that ethics "is really the crux of any professional organization . . . ".' ' . . . In a different contex, Dr. Wilder Penfield, moving a vote of thanks to Lord Adrian at Edinburgh in July, made an urgent plea that the medical profession should return to its old traditional behaviour. "We talk so much about medical science", he said, "that we forget to swear the Hippocratic Oath". "We never speak of our ethical code, and many who are not of us are ignorant of its existence".'

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